Hand: 10/61/91

=> d his full

(FILE 'HOME' ENTERED AT 12:27:40 ON 17 JUL 2006)

	FILE	'REGI	STRY' ENTERE		27:59 ON 17 JUL 2006
L1		1	SEA ABB=ON D SCA	PLU=ON	FERRIOXAMINE/CN
L2		. 1	SEA ABB=ON D SCA	PLU=ON	FERRIOXAMINE B/CN
L3		1	SEA ABB=ON	PLU=ON	FERRIOXAMINE B C?/CN
L4		1	SEA ABB=ON	PLU=ON	FERRIOXAMINE B H?/CN
L5		1	SEA ABB=ON	PLU=ON	· .
L6				PLU=ON	
L7		6		PLU=ON	
		_	E TRIHYDROX	AMI/CN	,
			E CP94/CN E CP 94/CN	, с.:	
L8		1		DI II-OM	CP 94/CN
по		1	D SCA E EDTA/CN	PLO=ON	CF 94/CN
L9		-	•	DIII ON	EDTA/CN
			D SCA	PLU=ON	
L10		1	SEA ABB=ON D SCA	PLU=ON	"EDTA (CHELATING AGENT)"/CN
L***	DEL	1	S L9-L10 E DEFEROX/C	N	
L11		1	SEA ABB=ON	PLU=ON	DEFEROXAMINE B MESYLATE/CN
L12		0	SEA ABB=ON	PLU=ON	L11 AND L7
L13		1	SEA ABB=ON E METHANESU	PLU=ON LFONATE"	("DEFEROXAMINE MESYLATE"/CN OR "DEFEROXAMIN/CN)
L14		0		PLU=ON	L13 AND L7
L15		1	SEA ABB=ON		DESFERAL/CN
		_	D SCA	120-011	
L16		1		PLU=ON	DESFERAL M?/CN
		_	E APOFERRIT		
L17		1	SEA ABB=ON E CDTA/CN		APOFERRITIN?/CN
L18		1		PLU=ON	CDTA/CN
			D SCA		•
			E DTPA/CN		
L19		1	SEA ABB=ON	PLU=ON	DTPA/CN
			E PENICILLA	MIN/CN	
L20		1	SEA ABB=ON	PLU=ON	PENICILLAMINE/CN
			D SCA		
			E BATHOCUPRO		
			E BATHOCUPPI		
L21		6	SEA ABB=ON		BATHOCUP?/CN
			E DIETHYLEN		·
L22			SEA ABB=ON		
L23		23	SEA ABB=ON		
					L11 OR L12 OR L13 OR L14 OR L15 OR L16 OR
				OR L19 0	R L20 OR L21 OR L22)
			D COST		

FILE 'STNGUIDE' ENTERED AT 12:42:46 ON 17 JUL 2006

FILE 'STNGUIDE' ENTERED AT 12:53:16 ON 17 JUL 2006 .

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FILE 'HCAPLUS' ENTERED AT 12:54:03 ON 17 JUL 2006
               E US2003-617943/APPS
              1 SEA ABB=ON PLU=ON US2003-617943/APPS
L24
               D SCA
               D IALL
    FILE 'STNGUIDE' ENTERED AT 12:54:39 ON 17 JUL 2006
     FILE 'HCAPLUS' ENTERED AT 12:57:00 ON 17 JUL 2006
                SEL RN
     FILE 'REGISTRY' ENTERED AT 12:57:13 ON 17 JUL 2006
             36 SEA ABB=ON PLU=ON (138-14-7/BI OR 70-51-9/BI OR 115900-75-9/B
L25
                I OR 117-39-5/BI OR 13291-61-7/BI OR 146426-40-6/BI OR
                14836-73-8/BI OR 153-18-4/BI OR 17912-87-7/BI OR 446-72-0/BI
                OR 480-16-0/BI OR 480-18-2/BI OR 480-40-0/BI OR 480-41-1/BI OR
                482-39-3/BI OR 490-46-0/BI OR 491-70-3/BI OR 491-80-5/BI OR
                50-78-2/BI OR 52-67-5/BI OR 520-26-3/BI OR 520-27-4/BI OR
                520-33-2/BI OR 520-36-5/BI OR 522-12-3/BI OR 525-82-6/BI OR
                577-85-5/BI OR 60-00-4/BI OR 67-43-6/BI OR 73348-75-1/BI OR
                7439-89-6/BI OR 7440-50-8/BI OR 7447-39-4/BI OR 75-91-2/BI OR
                7758-94-3/BI OR 989-51-5/BI)
              8 SEA ABB=ON PLU=ON L25 AND L23
L26
     FILE 'HCAPLUS' ENTERED AT 12:57:43 ON 17 JUL 2006
          37763 SEA ABB=ON PLU=ON L26
L27
              1 SEA ABB=ON PLU=ON L24 AND L27
L28
               D SCA
     FILE 'REGISTRY' ENTERED AT 12:58:24 ON 17 JUL 2006
              1 SEA ABB=ON PLU=ON 70-51-9
L29
     FILE 'HCAPLUS' ENTERED AT 12:58:37 ON 17 JUL 2006
           2619 SEA ABB=ON PLU=ON L29
L30
     FILE 'REGISTRY' ENTERED AT 12:58:52 ON 17 JUL 2006
             24 SEA ABB=ON PLU=ON L23 OR L29
L31
     FILE 'HCAPLUS' ENTERED AT 13:32:52 ON 17 JUL 2006
          40989 SEA ABB=ON PLU=ON L31
L32
              1 SEA ABB=ON PLU=ON L24 AND L32
L33
                D SCA
     FILE 'REGISTRY' ENTERED AT 13:33:45 ON 17 JUL 2006
             27 SEA ABB=ON PLU=ON L25 NOT L31
L34
     FILE 'HCAPLUS' ENTERED AT 13:34:06 ON 17 JUL 2006
          16008 SEA ABB=ON PLU=ON L34 (L) THU/RL
L35
              1 SEA ABB=ON PLU=ON L24 AND L35
L36
                D SCA
                SEL HIT RN
     FILE 'REGISTRY' ENTERED AT 13:35:00 ON 17 JUL 2006
             22 SEA ABB=ON PLU=ON (117-39-5/BI OR 146426-40-6/BI OR 153-18-4/
L37
                BI OR 17912-87-7/BI OR 446-72-0/BI OR 480-16-0/BI OR 480-18-2/B
                I OR 480-40-0/BI OR 480-41-1/BI OR 482-39-3/BI OR 490-46-0/BI
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525-82-6/BI OR 577-85-5/BI OR 989-51-5/BI)

OR 491-70-3/BI OR 491-80-5/BI OR 50-78-2/BI OR 520-26-3/BI OR 520-27-4/BI OR 520-33-2/BI OR 520-36-5/BI OR 522-12-3/BI OR

5.1

11/06/2006

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L38
              1 SEA ABB=ON PLU=ON 50-78-2
             21 SEA ABB=ON PLU=ON L37 NOT L38
L39
     FILE 'HCAPLUS' ENTERED AT 13:35:37 ON 17 JUL 2006
          34358 SEA ABB=ON PLU=ON L39
L40
              1 SEA ABB=ON PLU=ON L40 AND L24
L41
                D SCA
     FILE 'STNGUIDE' ENTERED AT 13:36:14 ON 17 JUL 2006
     FILE 'HCAPLUS' ENTERED AT 13:36:39 ON 17 JUL 2006
                E ATAXIA TELANGIECTASIA+ALL/CT
                E E2+ALL
           1665 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA/OBI
L42
           2356 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA/BI
L43
              O SEA ABB=ON PLU=ON LOUIS BAR/OBI
L44
              8 SEA ABB=ON PLU=ON LOUIS BAR/BI
L45
              8 SEA ABB=ON PLU=ON LOUIS-BAR/BI
L46
              O SEA ABB=ON PLU=ON CEREBELLO OCULOCUTANEOUS TELANGIECT?/BI
L47
              O SEA ABB=ON PLU=ON CEREBELLO OCULOT?/BI
L48
     FILE 'STNGUIDE' ENTERED AT 13:39:59 ON 17 JUL 2006
     FILE 'HCAPLUS' ENTERED AT 13:40:24 ON 17 JUL 2006
L49
           2363 SEA ABB=ON PLU=ON (ATAXIA (2A) TELANGIECT?)/BI
                E CHELATING AGENT+ALL/CT
                E CHELATING AGENTS+ALL/CT
L50
          15362 SEA ABB=ON PLU=ON CHELATING AGENTS+OLD, NT/CT
          40989 SEA ABB=ON PLU=ON L31
L51
           2369 SEA ABB=ON PLU=ON (L42 OR L43 OR L44 OR L45 OR L46 OR L47 OR
L52
                L48 OR L49)
L53
          52491 SEA ABB=ON PLU=ON (L50 OR L51)
              7 SEA ABB=ON PLU=ON L52 AND L53
L54
                D SCA
L55
          34358 SEA ABB=ON PLU=ON L39
         4 SEA ABB=ON PLU=ON L54 AND L55 144038 SEA ABB=ON PLU=ON ANTIOXID?/BI
L56
L57
                E FLAVANOIDS+ALL/CT
                E E2+ALL/CT
          57074 SEA ABB=ON PLU=ON FLAVONOIDS+NT,OLD,UF/CT 34491 SEA ABB=ON PLU=ON FLAV!NOID?/BI
L58
L59
L60
              4 SEA ABB=ON PLU=ON L54 AND (L57 OR L58 OR L59)
                D SCA
L61
          11651 SEA ABB=ON PLU=ON ?HYDROXAMIC ACID?/BI
                E FERRITINS+ALL/CT
L62
           9439 SEA ABB=ON PLU=ON FERRITINS+OLD, UF/CT
             17 SEA ABB=ON PLU=ON L52 AND (L61 OR L62)
14 SEA ABB=ON PLU=ON L63 NOT L54
L63
L64
L65
            374 SEA ABB=ON PLU=ON FERRITINS/CT (L) (THU OR BAC OR DMA OR PAC
                OR PKT)/RL
L66
              5 SEA ABB=ON PLU=ON L65 AND L52
L67
              3 SEA ABB=ON PLU=ON L66 NOT L54
                D SCA
                D SCA TI
                E HYDROXAMIC ACIDS+ALL/CT
L68
          15322 SEA ABB=ON PLU=ON HYDROXAMIC ACIDS+NT/CT
L69
             29 SEA ABB=ON PLU=ON L68 AND L52
L70
         132130 SEA ABB=ON PLU=ON CHELAT?/BI
L71
              3 SEA ABB=ON PLU=ON L69 AND L70
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D SCA

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6 SEA ABB=ON PLU=ON L70 AND L52
1 SEA ABB=ON PLU=ON L72 NOT L54
L72
L73
                     D SCA
             29887 SEA ABB=ON PLU=ON WANG S?/AU

49 SEA ABB=ON PLU=ON SHACKELFORD R?/AU

12 SEA ABB=ON PLU=ON SHACKLEFORD R?/AU

5 SEA ABB=ON PLU=ON L74 AND (L75 OR L76)

12 SEA ABB=ON PLU=ON L52 AND (L74 OR L75 OR L76)
L74
L75
L76
L77
L78
       FILE 'MEDLINE' ENTERED AT 14:04:08 ON 17 JUL 2006
                    D COST
               9295 SEA ABB=ON PLU=ON WANG S?/AU
81 SEA ABB=ON PLU=ON SHACKELFORD R?/AU
10 SEA ABB=ON PLU=ON SHACKLEFORD R?/AU
3 SEA ABB=ON PLU=ON L79 AND (L80 OR L81)
L79
L80
L81
                       E ATAXIA TELANGIECTASIA+ALL/CT
              2457 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA/CT
L*** DEL 3951 S ATAXIA TELANGIECTAS?
                      D TRIAL 1-3
               3932 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA
3935 SEA ABB=ON PLU=ON ATAXIA (2A) TELANGIECTASIA
3935 SEA ABB=ON PLU=ON (L83 OR L84 OR L85)
13 SEA ABB=ON PLU=ON L86 AND (L79 OR L80 OR L81)
L85
L86
                       E CHELATING AGENTS+ALL/CT
       13204 SEA ABB=ON PLU=ON CHELATING AGENTS/CT
92986 SEA ABB=ON PLU=ON CHELATING AGENTS+NT/CT
3231 SEA ABB=ON PLU=ON IRON CHELATING AGENTS/CT
19528 SEA ABB=ON PLU=ON IRON CHELATING AGENTS+NT/CT
L88
L89
L90
                      E IRON CHELATING AGENTS+ALL/CT
                   0 S SIDEPHORES/CT
L*** DEL
                0 S SIDEPHORES+NT/CT
L*** DEL
L92 1267 SEA ABB=ON PLU=ON SIDEROPHORES/CT
L93 6055 SEA ABB=ON PLU=ON SIDEROPHORES+NT/CT
       FILE 'REGISTRY' ENTERED AT 14:12:03 ON 17 JUL 2006
                       SET SMARTSELECT ON
                       SEL PLU=ON L31 1- CHEM : 255 TERMS
L94
                       SET SMARTSELECT OFF
       FILE 'MEDLINE' ENTERED AT 14:12:07 ON 17 JUL 2006
              68933 SEA ABB=ON PLU=ON L94
L95
                    7 SEA ABB=ON PLU=ON L86 AND ((L88 OR L89 OR L90 OR L91 OR L92
L96
                       OR L93) OR L95)
                       D TRIAL 1-7
              37091 SEA ABB=ON PLU=ON CHELAT?
              5 SEA ABB=ON PLU=ON L86 AND L97
0 SEA ABB=ON PLU=ON L98 NOT L96
61053 SEA ABB=ON PLU=ON ANTIOXID?
              18939 SEA ABB=ON PLU=ON FLAV!NOID?/BI
              32882 SEA ABB=ON PLU=ON FLAVONOIDS+NT/CT
                    0 S FLAVANOIDS+NT/CT
L*** DEL
                    QUE ABB=ON PLU=ON TRANSITION ELEMENTS+NT/CT
3 SEA ABB=ON PLU=ON L96 AND ((L100 OR L101 OR L102))
                      D TRIAL 1-3
                    3 SEA ABB=ON PLU=ON L96 AND ((L100 OR L101 OR L102 OR L103))
L105
                       D TRIAL 1-3
       FILE 'REGISTRY' ENTERED AT 14:18:45 ON 17 JUL 2006
                      SET SMARTSELECT ON
                       SEL PLU=ON L39 1- CHEM: 344 TERMS
L106
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SET SMARTSELECT OFF

mires 0

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FILE 'MEDLINE' ENTERED AT 14:18:48 ON 17 JUL 2006
           19788 SEA ABB=ON PLU=ON L106
L108
                2 SEA ABB=ON PLU=ON L107 AND L96
                  D TRIAL 1-2
L109
                   OUE ABB=ON PLU=ON FERRIOXAMIN? OR DEFEROXAMIN? OR DESFERROXAM
                  IN? OR DEFERRIOXAMIN?
                  OUE ABB=ON PLU=ON EDETIC ACID/CT
L110
                  OUE ABB=ON PLU=ON CP94
L111
                 OUE ABB=ON PLU=ON HYDROXAMIC ACIDS/CT
L112
                 QUE ABB=ON PLU=ON APOFERRITIN/CT
L113
                 QUE ABB=ON PLU=ON CDTA
L114
                QUE ABB=ON PLU=ON DTPA OR PENTATIC ACID
QUE ABB=ON PLU=ON PENICILLAMINE
QUE ABB=ON PLU=ON BATHOCUPROINE
QUE ABB=ON PLU=ON BATHOCUPROIN
L115
L116
L117
L118
              6 SEA ABB=ON PLU=ON L86 AND (L109 OR L110 OR L111 OR L112 OR
L119
                  L113 OR L114 OR L115 OR L116 OR L117 OR L118)
                  D TRIAL 1-6
                1 SEA ABB=ON PLU=ON L119 AND L107
L120
                  D TRIAL
               13 SEA ABB=ON PLU=ON L82 OR L87
T-121
               10 SEA ABB=ON PLU=ON L96 OR L98 OR L105 OR L108 OR L119 OR L120
T:122
               7 SEA ABB=ON PLU=ON L122 NOT L121
1.123
     FILE 'EMBASE' ENTERED AT 14:31:14 ON 17 JUL 2006
             7035 SEA ABB=ON PLU=ON WANG S?/AU
L124
             5 SEA ABB=ON PLU=ON SHACKLEFORD R?/AU
L125
               27 SEA ABB=ON PLU=ON SHACKELFORD R?/AU
L126
               4 SEA ABB=ON PLU=ON L124 AND (L125 OR L126)
L127
                  E ATAXIA TELANGIECTASIA/CT
                  E ATAXIA TELANGIECTASIA+ALL/CT
                  E ATAXIA TELANGIECTASIA+UF/CT
          2332 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA+UF/CT
3044 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA
62 SEA ABB=ON PLU=ON LOUIS BAR
2 SEA ABB=ON PLU=ON ATAXIA TELANGIECTATICA
0 SEA ABB=ON PLU=ON TELANGIECTASIA CEREBELLOOCULOCUTANEA
0 SEA ABB=ON PLU=ON TELANGIECTASIA CEREBELLO OCULOCUTANEA
3053 SEA ABB=ON PLU=ON (L128 OR L129 OR L130 OR L131 OR L132 OR
L128
L129
L130
L131
L132
L133
L134
                  L133)
L135
               11 SEA ABB=ON PLU=ON (L124 OR L125 OR L126) AND L134
                  E CHELATING AGENT+ALL/CT
L136 98621 SEA ABB=ON PLU=ON CHELATING AGENT+NT/CT
     FILE 'REGISTRY' ENTERED AT 14:35:53 ON 17 JUL 2006
                  SET SMARTSELECT ON
                  SEL PLU=ON L31 1- CHEM: 255 TERMS
L137
                  SET SMARTSELECT OFF
      FILE 'EMBASE' ENTERED AT 14:35:55 ON 17 JUL 2006
           61283 SEA ABB=ON PLU=ON L137
14 SEA ABB=ON PLU=ON L134 AND (L136 OR L138)
L138
L139
                  D TRIAL 1-14
                  E FLAVONOID+ALL/CT
L140
           25033 SEA ABB=ON PLU=ON FLAVONOID+NT/CT
                  E ANTIOXIDANT+ALL/CT
L141
           35447 SEA ABB=ON PLU=ON ANTIOXIDANT+NT/CT
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4 SEA ABB=ON PLU=ON L139 AND (L140 OR L141)
L142
                 D TRIAL 1-4
    FILE 'REGISTRY' ENTERED AT 14:41:15 ON 17 JUL 2006
                 SET SMARTSELECT ON
                  SEL PLU=ON L39 1- CHEM: 344 TERMS
L143
                  SET SMARTSELECT OFF
    FILE 'EMBASE' ENTERED AT 14:41:16 ON 17 JUL 2006
L144 24101 SEA ABB=ON PLU=ON L143
L145 2 SEA ABB=ON PLU=ON L139 AND L144
                 D TRIAL
        14 SEA ABB=ON PLU=ON L139 OR L142 OR L145
11 SEA ABB=ON PLU=ON L127 OR L135
10 SEA ABB=ON PLU=ON L146 NOT L147
                 D TRIAL 2
L146
L147
L148
               D TRIAL 1-5
L149 31221 SEA ABB=ON PLU=ON CHELAT?
L150 4 SEA ABB=ON PLU=ON L149 AND L134
L150
                 D TRIAL
                  D TRIAL 2-4
    FILE 'BIOSIS' ENTERED AT 14:47:22 ON 17 JUL 2006
L*** DEL 4 S WANS S?/AU
          10521 SEA ABB=ON PLU=ON WANG S?/AU

52 SEA ABB=ON PLU=ON SHACKELFORD R?/AU

7 SEA ABB=ON PLU=ON SHACKLEFORD R?/AU

6 SEA ABB=ON PLU=ON L151 AND (L152 OR L153)

3180 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA

18 SEA ABB=ON PLU=ON (L151 OR L152 OR L153) AND L155
L151
L152
L153 .
L154
L155
L156
                 E CHELATING AGENTS+ALL/CT
                  E E3+ALL
L157 38028 SEA ABB=ON PLU=ON CHELAT?
    FILE 'REGISTRY' ENTERED AT 14:50:27 ON 17 JUL 2006
             SET SMARTSELECT ON
                 SEL PLU=ON L31 1- CHEM: 255 TERMS
L158
                  SET SMARTSELECT OFF
  FILE 'BIOSIS' ENTERED AT 14:50:28 ON 17 JUL 2006
D SCA
           82137 SEA ABB=ON PLU=ON ANTIOXID? OR FLAV!NOID?
L169
     FILE 'REGISTRY' ENTERED AT 14:55:16 ON 17 JUL 2006
                 SET SMARTSELECT ON
                  SEL PLU=ON L39 1- CHEM : 344 TERMS
T.170
                  SET SMARTSELECT OFF
    FILE 'BIOSIS' ENTERED AT 14:55:17 ON 17 JUL 2006
L171 31206 SEA ABB=ON PLU=ON L170
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3 SEA ABB=ON PLU=ON L171 AND L168
L172
                 D SCA
               O SEA ABB=ON PLU=ON L168 AND (L161 OR L162)
L173
              18 SEA ABB=ON PLU=ON L154 OR L156
L174
              76 S L168 OR L172 OR L!73
L*** DEL
              7 SEA ABB=ON PLU=ON L168 OR L172 OR L173
L175
               4 SEA ABB=ON PLU=ON L174 AND L175
L176
                 D SCA
     FILE 'EMBASE' ENTERED AT 14:58:18 ON 17 JUL 2006
             14 SEA ABB=ON PLU=ON L146 OR L150
           4 SEA ABB=ON PLU=ON L147 AND L177
L178
     FILE 'MEDLINE' ENTERED AT 14:59:22 ON 17 JUL 2006
               3 SEA ABB=ON PLU=ON L121 AND L122
     FILE 'HCAPLUS' ENTERED AT 15:00:01 ON 17 JUL 2006
              12 SEA ABB=ON PLU=ON (L77 OR L78)
7 SEA ABB=ON PLU=ON L54 OR L56 OR L71
L180
L181
               4 SEA ABB=ON PLU=ON L180 AND L181
L182
     FILE 'STNGUIDE' ENTERED AT 15:00:33 ON 17 JUL 2006
     FILE 'USPATFULL' ENTERED AT 15:01:27 ON 17 JUL 2006
           5870 SEA ABB=ON PLU=ON L31
2355 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA
6 SEA ABB=ON PLU=ON L183 AND L184
L183
L184
L185
                 D SCA
               0 S L31 (L) THU/RL
L*** DEL
                 D KWIC 1-6
           1795 SEA ABB=ON PLU=ON L39
L186
               1 SEA ABB=ON PLU=ON L185 AND L186
L187
     FILE 'WPIX' ENTERED AT 15:05:21 ON 17 JUL 2006
           247 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA/BIX
1.188
           5402 SEA ABB=ON PLU=ON WANG S?/AU

0 SEA ABB=ON PLU=ON SHACKLEFORD R?/AU

3 SEA ABB=ON PLU=ON SHACKELFORD R?/AU

1 SEA ABB=ON PLU=ON L189 AND (L190 OR L191)
L189
L190
L191
L192
                 D SCA
                 SEL DCN
               O SEA ABB=ON PLU=ON (RAAMBT/DCR OR RAGNO8/DCR OR RAODFA/DCR OR
L193
                 RAOEMC/DCR OR RAOJBK/DCR OR RAOOTF/DCR OR RAOO55/DCR OR
                 RA021P/DCR OR RA0529/DCR OR RA1HHQ/DCR OR RA1XA5/DCR OR
                 RA37W9/DCR OR R00064/DCR OR R00195/DCR OR R00268/DCR OR
                 R00971/DCR OR R01179/DCR OR R01318/DCR OR R01319/DCR OR
                 R03811/DCR OR R03812/DCR OR R03949/DCR OR R04870/DCR OR
                 R06069/DCR OR R06174/DCR OR R06413/DCR OR R06747/DCR OR
                 R07001/DCR OR R07027/DCR OR R08105/DCR OR R08504/DCR OR
                 R09163/DCR OR R09222/DCR OR R09884/DCR OR R11605/DCR OR
                 R19085/DCR OR R19452/DCR OR R20811/DCR OR R22037/DCR)
L194
               O SEA ABB=ON PLU=ON (RAAMBT/DCRE OR RAGNQ8/DCRE OR RAODFA/DCRE
                 OR RAOEMC/DCRE OR RAOJBK/DCRE OR RAOOTF/DCRE OR RAOO55/DCRE OR
                 RA021P/DCRE OR RA0529/DCRE OR RA1HHQ/DCRE OR RA1XA5/DCRE OR
                 RA37W9/DCRE OR R00064/DCRE OR R00195/DCRE OR R00268/DCRE OR
                 R00971/DCRE OR R01179/DCRE OR R01318/DCRE OR R01319/DCRE OR
                 R03811/DCRE OR R03812/DCRE OR R03949/DCRE OR R04870/DCRE OR
                 R06069/DCRE OR R06174/DCRE OR R06413/DCRE OR R06747/DCRE OR
                 R07001/DCRE OR R07027/DCRE OR R08105/DCRE OR R08504/DCRE OR
                 R09163/DCRE OR R09222/DCRE OR R09884/DCRE OR R11605/DCRE OR
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R19085/DCRE OR R19452/DCRE OR R20811/DCRE OR R22037/DCRE)

FILE 'STNGUIDE' ENTERED AT 15:09:00 ON 17 JUL 2006

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FILE 'WPIX' ENTERED AT 15:09:49 ON 17 JUL 2006
            5533 SEA ABB=ON PLU=ON (RAAMBT/DCN OR RAGNQ8/DCN OR RAODFA/DCN OR
L195
                 RAOEMC/DCN OR RAOJBK/DCN OR RAOOTF/DCN OR RAOO55/DCN OR
                 RA021P/DCN OR RA0529/DCN OR RA1HHQ/DCN OR RA1XA5/DCN OR
                 RA37W9/DCN OR R00064/DCN OR R00195/DCN OR R00268/DCN OR
                 R00971/DCN OR R01179/DCN OR R01318/DCN OR R01319/DCN OR
                 R03811/DCN OR R03812/DCN OR R03949/DCN OR R04870/DCN OR
                 R06069/DCN OR R06174/DCN OR R06413/DCN OR R06747/DCN OR
                 R07001/DCN OR R07027/DCN OR R08105/DCN OR R08504/DCN OR
                 R09163/DCN OR R09222/DCN OR R09884/DCN OR R11605/DCN OR
                 R19085/DCN OR R19452/DCN OR R20811/DCN OR R22037/DCN)
               0 SEA ABB=ON PLU=ON L188 AND (L189 OR L190 OR L191)
2 SEA ABB=ON PLU=ON L188 AND L195
L196
L197
                 D SCA
                 D HIT L197 1-2
                 SEL HIT DCN
             623 SEA ABB=ON PLU=ON (R06069-/DCN OR R00971-/DCN OR RA0055-/DCN
L198
                 OR R06069/DCN OR RA0055/DCN)
               0 SEA ABB=ON PLU=ON (R06069-/DRN OR R00971-/DRN OR RA0055-/DRN
L199
                 OR R06069/DRN OR RA0055/DRN)
               O SEA ABB=ON PLU=ON (R06069-/SDCE OR R00971-/SDCE OR RA0055-/SD
L200
                 CE OR R06069/SDCE OR RA0055/SDCE)
               O SEA ABB=ON PLU=ON (R06069-/SDRN OR R00971-/SDRN OR RA0055-/SD
L201
                 RN OR R06069/SDRN OR RA0055/SDRN)
     FILE 'STNGUIDE' ENTERED AT 15:14:29 ON 17 JUL 2006
     FILE 'USPATFULL' ENTERED AT 15:14:54 ON 17 JUL 2006
            1992 SEA ABB=ON PLU=ON WANG S?/AU
5 SEA ABB=ON PLU=ON SHACKELFORD R?/AU
3 SEA ABB=ON PLU=ON SHACKLEFORD R?/AU
1 SEA ABB=ON PLU=ON L202 AND (L203 OR L204)
1 SEA ABB=ON PLU=ON (L202 OR L203 OR L204) AND (L185 OR L187)
L202
L203
L204
L205
L206
     FILE 'WPIX' ENTERED AT 15:15:49 ON 17 JUL 2006
               0 SEA ABB=ON PLU=ON L197 AND ((L189 OR L190 OR L191))
L207
     FILE 'STNGUIDE' ENTERED AT 15:16:36 ON 17 JUL 2006
                 D COST
     FILE 'STNGUIDE' ENTERED AT 15:16:46 ON 17 JUL 2006
     FILE 'REGISTRY' ENTERED AT 15:22:10 ON 17 JUL 2006
     FILE 'HCAPLUS' ENTERED AT 15:22:11 ON 17 JUL 2006
                 D QUE L77
                 D QUE L78
                 D QUE L182
              12 SEA ABB=ON PLU=ON (L77 OR L78) OR L182
L208
     FILE 'MEDLINE' ENTERED AT 15:22:15 ON 17 JUL 2006
                 D QUE L82
                 D OUE L87
                 D OUE L179
L209
              13 SEA ABB=ON PLU=ON L82 OR L87 OR L179
```

ψ.

```
23 3 36600
    FILE 'EMBASE' ENTERED AT 15:22:20 ON 17 JUL 2006
               D OUE L127
               D QUE L135
               D QUE L178
             11 SEA ABB=ON PLU=ON L127 OR L135 OR L178
L210
    FILE 'BIOSIS' ENTERED AT 15:22:24 ON 17 JUL 2006
               D QUE L154
               D QUE L156
               D QUE L176
             18 SEA ABB=ON PLU=ON L154 OR L156 OR L176
L211
    FILE 'USPATFULL' ENTERED AT 15:22:28 ON 17 JUL 2006
               D QUE L205
               D QUE L206
              1 SEA ABB=ON PLU=ON L205 OR L206
L212
    FILE 'WPIX' ENTERED AT 15:22:31 ON 17 JUL 2006
               D QUE L192
               D QUE L196
               D QUE L207
              1 SEA ABB=ON PLU=ON L192 OR L196 OR L207
L213
     FILE 'HCAPLUS, MEDLINE, EMBASE, BIOSIS, USPATFULL, WPIX' ENTERED AT
     15:23:43 ON 17 JUL 2006
            22 DUP REM L208 L209 L210 L211 L212 L213 (34 DUPLICATES REMOVED)
L214
                    ANSWERS '1-12' FROM FILE HCAPLUS
                    ANSWERS '13-15' FROM FILE MEDLINE
                    ANSWERS '16-22' FROM FILE BIOSIS
    FILE 'STNGUIDE' ENTERED AT 15:23:57 ON 17 JUL 2006
     FILE 'HCAPLUS, MEDLINE, BIOSIS' ENTERED AT 15:24:47 ON 17 JUL 2006
               D IBIB ABS HITIND HITSTR L214 1-12
     FILE 'STNGUIDE' ENTERED AT 15:24:50 ON 17 JUL 2006
    FILE 'HCAPLUS, MEDLINE, BIOSIS' ENTERED AT 15:25:01 ON 17 JUL 2006
               D IALL L214 13-22
     FILE 'STNGUIDE' ENTERED AT 15:25:02 ON 17 JUL 2006
     FILE 'STNGUIDE' ENTERED AT 15:25:52 ON 17 JUL 2006
     FILE 'HCAPLUS' ENTERED AT 15:30:14 ON 17 JUL 2006
               D QUE L54
                D QUE L56
               D QUE L71
L215
              3 SEA ABB=ON PLU=ON (L54 OR L56 OR L71) NOT L208
     FILE 'MEDLINE' ENTERED AT 15:30:19 ON 17 JUL 2006
                D QUE L96
                D QUE L98
                D QUE L105
                D OUE L108
```

FILE 'EMBASE' ENTERED AT 15:30:24 ON 17 JUL 2006

D OUE L119

L209

L216

7 SEA ABB=ON PLU=ON (L96 OR L98 OR L105 OR L108 OR L119) NOT

D QUE L139 D QUE L142 D QUE L145 D QUE L150

10 SEA ABB=ON PLU=ON (L139 OR L142 OR L145 OR L150) NOT L210 L217

FILE 'BIOSIS' ENTERED AT 15:30:29 ON 17 JUL 2006

D QUE L168 D QUE L172

D QUE L173

3 SEA ABB=ON PLU=ON (L168 OR L172 OR L173) NOT L211 L218

FILE 'USPATFULL' ENTERED AT 15:30:33 ON 17 JUL 2006

D OUE L185

D QUE L187

5 SEA ABB=ON PLU=ON (L185 OR L187) NOT L212 L219

FILE 'WPIX' ENTERED AT 15:30:37 ON 17 JUL 2006

D QUE L197

2 SEA ABB=ON PLU=ON L197 NOT L213 L220

FILE 'STNGUIDE' ENTERED AT 15:31:04 ON 17 JUL 2006

FILE 'HCAPLUS, MEDLINE, EMBASE, BIOSIS, USPATFULL, WPIX' ENTERED AT 15:31:21 ON 17 JUL 2006

24 DUP REM L215 L216 L217 L218 L219 L220 (6 DUPLICATES REMOVED) L221

ANSWERS '1-3' FROM FILE HCAPLUS ANSWERS '4-9' FROM FILE MEDLINE ANSWERS '10-16' FROM FILE EMBASE ANSWER '17' FROM FILE BIOSIS

ANSWERS '18-22' FROM FILE USPATFULL

ANSWERS '23-24' FROM FILE WPIX

D IBIB ABS HITIND HITSTR L221 1-3

D IALL L221 4-17

D IBIB ABS KWIC HITSTR L221 18-22

D IALL IND L221 23-24

FILE HOME

FILE REGISTRY

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

14 JUL 2006 HIGHEST RN 892755-86-1 STRUCTURE FILE UPDATES: DICTIONARY FILE UPDATES: 14 JUL 2006 HIGHEST RN 892755-86-1

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http://www.cas.org/ONLINE/UG/regprops.html

FILE STNGUIDE

FILE CONTAINS CURRENT INFORMATION.

LAST RELOADED: Jul 17, 2006 (20060717/UP).

13

FILE HCAPLUS

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FILE COVERS 1907 - 17 Jul 2006 VOL 145 ISS 4 FILE LAST UPDATED: 16 Jul 2006 (20060716/ED)

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This file contains CAS Registry Numbers for easy and accurate substance identification.

FILE MEDLINE

FILE LAST UPDATED: 15 JUL 2006 (20060715/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 is now (26 Feb.) available. For details on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>). See also:

http://www.nlm.nih.gov/mesh/

http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html

http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html

http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

FILE EMBASE

FILE COVERS 1974 TO 17 Jul 2006 (20060717/ED)

EMBASE has been reloaded. Enter HELP RLOAD for details.

EMBASE is now updated daily. SDI frequency remains weekly (default) and biweekly.

This file contains CAS Registry Numbers for easy and accurate substance identification.

FILE BIOSIS

FILE COVERS 1969 TO DATE.

CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 12 July 2006 (20060712/ED)

FILE USPATFULL

FILE COVERS 1971 TO PATENT PUBLICATION DATE: 13 Jul 2006 (20060713/PD)

FILE LAST UPDATED: 13 Jul 2006 (20060713/ED)

HIGHEST GRANTED PATENT NUMBER: US7076805

HIGHEST APPLICATION PUBLICATION NUMBER: US2006156447

CA INDEXING IS CURRENT THROUGH 11 Jul 2006 (20060711/UPCA)

ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 13 Jul 2006 (20060713/PD)

REVISED CLASS FIELDS (/NCL) LAST RELOADED: Feb 2006

USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Feb 2006

FILE WPIX

FILE LAST UPDATED: 14 JUL 2006 <20060714/UP>

MOST RECENT DERWENT UPDATE: 200645 <200645/DW>

DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

>>> FOR A COPY OF THE DERWENT WORLD PATENTS INDEX STN USER GUIDE,
 PLEASE VISIT:
http://www.stn-international.de/training_center/patents/stn_guide.pdf <</pre>

>>> FOR DETAILS OF THE PATENTS COVERED IN CURRENT UPDATES, SEE http://scientific.thomson.com/support/patents/coverage/latestupdates/

>>> PLEASE BE AWARE OF THE NEW IPC REFORM IN 2006, SEE http://www.stn-international.de/stndatabases/details/ipc_reform.html and http://scientific.thomson.com/media/scpdf/ipcrdwpi.pdf <<<

>>> FOR FURTHER DETAILS ON THE FORTHCOMING DERWENT WORLD PATENTS
INDEX ENHANCEMENTS PLEASE VISIT:
http://www.stn-international.de/stndatabases/details/dwpi_r.html <<<

=>

=> file registry
FILE 'REGISTRY' ENTERED AT 15:22:10 ON 17 JUL 2006
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STRUCTURE FILE UPDATES: 14 JUL 2006 HIGHEST RN 892755-86-1 DICTIONARY FILE UPDATES: 14 JUL 2006 HIGHEST RN 892755-86-1

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TSCA INFORMATION NOW CURRENT THROUGH January 6, 2006

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REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

=> file hcaplus

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FILE COVERS 1907 - 17 Jul 2006 VOL 145 ISS 4 FILE LAST UPDATED: 16 Jul 2006 (20060716/ED)

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This file contains CAS Registry Numbers for easy and accurate substance identification.
'OBI' IS DEFAULT SEARCH FIELD FOR 'HCAPLUS' FILE

=> d que L77

L74	29887	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	WANG S?/AU
L75	49	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	SHACKELFORD R?/AU
L76	12	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	SHACKLEFORD R?/AU
L77	5	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	L74 AND (L75 OR L76)

```
=> d que 178
          1665 SEA FILE=HCAPLUS ABB=ON PLU=ON ATAXIA TELANGIECTASIA/OBI
          2356 SEA FILE=HCAPLUS ABB=ON PLU=ON
                                               ATAXIA TELANGIECTASIA/BI
L43
            O SEA FILE=HCAPLUS ABB=ON PLU=ON
                                               LOUIS BAR/OBI
L44
             8 SEA FILE=HCAPLUS ABB=ON PLU=ON
                                               LOUIS BAR/BI
L45
                                               LOUIS-BAR/BI
             8 SEA FILE=HCAPLUS ABB=ON PLU=ON
L46
             O SEA FILE=HCAPLUS ABB=ON PLU=ON CEREBELLO OCULOCUTANEOUS
L47
               TELANGIECT?/BI
                                       PLU=ON CEREBELLO OCULOT?/BI
             O SEA FILE=HCAPLUS ABB=ON
L48
          2363 SEA FILE=HCAPLUS ABB=ON
                                       PLU=ON
                                               (ATAXIA (2A) TELANGIECT?)/BI
L49
          2369 SEA FILE=HCAPLUS ABB=ON PLU=ON
                                               (L42 OR L43 OR L44 OR L45 OR
L52
               L46 OR L47 OR L48 OR L49)
         29887 SEA FILE=HCAPLUS ABB=ON PLU=ON WANG S?/AU
L74
                                       PLU=ON
                                               SHACKELFORD R?/AU
            49 SEA FILE=HCAPLUS ABB=ON
L75
                                       PLU=ON SHACKLEFORD R?/AU
            12 SEA FILE=HCAPLUS ABB=ON
L76
            12 SEA FILE=HCAPLUS ABB=ON PLU=ON L52 AND (L74 OR L75 OR L76)
L78
=> d que L182
             1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
L1
             1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
L2
             1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
L3
             1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
L4
             1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
L5
             2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
L6
             6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L7
               OR L6)
             1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
L8
             1 SEA FILE=REGISTRY ABB=ON PLU=ON
                                                EDTA/CN
L9
                                                 "EDTA (CHELATING AGENT) "/CN
             1 SEA FILE=REGISTRY ABB=ON PLU=ON
L10
             1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN
L11
             O SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
L12
             1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
L13
               OR "DEFEROXAMINE METHANESULFONATE"/CN)
             O SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
L14
             1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
L15
             1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
L16
            1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
L17
            1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
L18
            1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
L19
             1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
L20
             6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
L21
             4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
L22
                C?/CN
            23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L23
               OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
                L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
             1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
L29
             24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
L31
            22 SEA FILE=REGISTRY ABB=ON PLU=ON (117-39-5/BI OR 146426-40-6/B
L37
                I OR 153-18-4/BI OR 17912-87-7/BI OR 446-72-0/BI OR 480-16-0/BI
                OR 480-18-2/BI OR 480-40-0/BI OR 480-41-1/BI OR 482-39-3/BI
                OR 490-46-0/BI OR 491-70-3/BI OR 491-80-5/BI OR 50-78-2/BI OR
                520-26-3/BI OR 520-27-4/BI OR 520-33-2/BI OR 520-36-5/BI OR
                522-12-3/BI OR 525-82-6/BI OR 577-85-5/BI OR 989-51-5/BI)
             1 SEA FILE=REGISTRY ABB=ON PLU=ON 50-78-2
L38
             21 SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38
L39
```

```
PLU=ON
                                                 ATAXIA TELANGIECTASIA/OBI
           1665 SEA FILE=HCAPLUS ABB=ON
                                                 ATAXIA TELANGIECTASIA/BI
                                         PLU=ON
           2356 SEA FILE=HCAPLUS ABB=ON
             O SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
                                                 LOUIS BAR/OBI
              8 SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
                                                 LOUIS BAR/BI
                                                 LOUIS-BAR/BI
              8 SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
                                                 CEREBELLO OCULOCUTANEOUS
              O SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
                TELANGIECT?/BI
                                                 CEREBELLO OCULOT?/BI
              O SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
           2363 SEA FILE=HCAPLUS ABB=ON
                                                  (ATAXIA (2A) TELANGIECT?)/BI
                                         PLU=ON
          15362 SEA FILE=HCAPLUS ABB=ON
                                                 CHELATING AGENTS+OLD, NT/CT
                                         PLU=ON
                                         PLU=ON
          40989 SEA FILE=HCAPLUS ABB=ON
                                        PLU=ON
                                                 (L42 OR L43 OR L44 OR L45 OR
           2369 SEA FILE=HCAPLUS ABB=ON
                L46 OR L47 OR L48 OR L49)
                                         PLU=ON
                                                  (L50 OR L51)
          52491 SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
                                                 L52 AND L53
              7 SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
                                                 L39
          34358 SEA FILE=HCAPLUS ABB=ON
              4 SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
                                                 L54 AND L55
          15322 SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
                                                 HYDROXAMIC ACIDS+NT/CT
             29 SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
                                                 L68 AND L52
         132130 SEA FILE=HCAPLUS ABB=ON
                                                 CHELAT?/BI
                                         PLU=ON
                                                 L69 AND L70
              3 SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
          29887 SEA FILE=HCAPLUS ABB=ON
                                                 WANG S?/AU
                                         PLU=ON
L74
             49 SEA FILE=HCAPLUS ABB=ON
                                                 SHACKELFORD R?/AU
                                         PLU=ON
L75
             12 SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
                                                 SHACKLEFORD R?/AU
L76
                                                 L74 AND (L75 OR L76)
             5 SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
L77
                                                 L52 AND (L74 OR L75 OR L76)
             12 SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
L78
                                                 (L77 OR L78)
             12 SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
L180
                                                 L54 OR L56 OR L71
              7 SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
L181
              4 SEA FILE=HCAPLUS ABB=ON
                                         PLU=ON
                                                 L180 AND L181
L182
```

=> s L77-L78 or L182

L208 12 (L77 OR L78) OR L182

=> file medline

FILE 'MEDLINE' ENTERED AT 15:22:15 ON 17 JUL 2006

FILE LAST UPDATED: 15 JUL 2006 (20060715/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 is now (26 Feb.) available. For details on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>). See also:

http://www.nlm.nih.gov/mesh/

http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html

http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html

http://www.nlm.nih.gov/pubs/techbull/nd05/nd05 2006 MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

```
=> d que L82
                         9295 SEA FILE=MEDLINE ABB=ON PLU=ON WANG S?/AU
81 SEA FILE=MEDLINE ABB=ON PLU=ON SHACKLEFORD R?/AU
10 SEA FILE=MEDLINE ABB=ON PLU=ON SHACKLEFORD R?/AU
3 SEA FILE=MEDLINE ABB=ON PLU=ON L79 AND (L80 OR L81)
L79
L80
L81
L82
=> d que L87
                         9295 SEA FILE=MEDLINE ABB=ON PLU=ON WANG S?/AU

81 SEA FILE=MEDLINE ABB=ON PLU=ON SHACKELFORD R?/AU

10 SEA FILE=MEDLINE ABB=ON PLU=ON SHACKLEFORD R?/AU

2457 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA/CT

3932 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA

3935 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA (2A) TELANGIECTASIA

3935 SEA FILE=MEDLINE ABB=ON PLU=ON (L83 OR L84 OR L85)

13 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND (L79 OR L80 OR L81)
L79
L80
L81
L83
L84
L85
L86
L87
=> d que 1179
                                 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L1
L2
L3
L4
L5
L6
L7
                                      OR L6)
                            1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT)"/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN
0 SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
0 PREFEROYAMINE METHANICAL TOWN TOWN
L8
L9
L10
L11
L12
L13
                                      OR "DEFEROXAMINE METHANESULFONATE"/CN)
                          OR "DEFEROXAMINE METHANESULFONATE"/CN)

O SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7

1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN

6 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI

C2/CN
L14
L15
L16
L17
L18
L19
L20
L21
L22
                                       C?/CN
                                23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L23
                                       OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
                                       L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
                                1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
22 SEA FILE=REGISTRY ABB=ON PLU=ON (117-39-5/BI OR 146426-40-6/B
L29
L31
L37
                                        I OR 153-18-4/BI OR 17912-87-7/BI OR 446-72-0/BI OR 480-16-0/BI
                                          OR 480-18-2/BI OR 480-40-0/BI OR 480-41-1/BI OR 482-39-3/BI
                                        OR 490-46-0/BI OR 491-70-3/BI OR 491-80-5/BI OR 50-78-2/BI OR
                                        520-26-3/BI OR 520-27-4/BI OR 520-33-2/BI OR 520-36-5/BI OR
                                        522-12-3/BI OR 525-82-6/BI OR 577-85-5/BI OR 989-51-5/BI)
                                1 SEA FILE=REGISTRY ABB=ON PLU=ON 50-78-2
21 SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38
 L38
 L39
```

200

```
9295 SEA FILE=MEDLINE ABB=ON PLU=ON WANG S?/AU
L79
               81 SEA FILE=MEDLINE ABB=ON PLU=ON SHACKELFORD R?/AU
L80
                10 SEA FILE=MEDLINE ABB=ON PLU=ON SHACKLEFORD R?/AU
L81
                3 SEA FILE=MEDLINE ABB=ON PLU=ON L79 AND (L80 OR L81)
L82
             3 SEA FILE=MEDLINE ABB=ON PLU=ON L/9 AND (L80 OR L81)

2457 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA/CT

3932 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA

3935 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA (2A) TELANGIECTASIA

3935 SEA FILE=MEDLINE ABB=ON PLU=ON (L83 OR L84 OR L85)

13 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND (L79 OR L80 OR L81)
L83
L84
L85
L86
L87
            13 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND (L79 OR L80 OR L81)
13204 SEA FILE=MEDLINE ABB=ON PLU=ON CHELATING AGENTS/CT
92986 SEA FILE=MEDLINE ABB=ON PLU=ON IRON CHELATING AGENTS+NT/CT
19528 SEA FILE=MEDLINE ABB=ON PLU=ON IRON CHELATING AGENTS+NT/CT
1267 SEA FILE=MEDLINE ABB=ON PLU=ON SIDEROPHORES/CT
6055 SEA FILE=MEDLINE ABB=ON PLU=ON SIDEROPHORES+NT/CT
L88
L89
L90
L91
L92
L93
                    SEL PLU=ON L31 1- CHEM : 255 TERMS
L94
            68933 SEA FILE=MEDLINE ABB=ON PLU=ON L94
L95
                 7 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND ((L88 OR L89 OR L90
L96
                    OR L91 OR L92 OR L93) OR L95)
            37091 SEA FILE=MEDLINE ABB=ON PLU=ON CHELAT?
L97
                 5 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND L97
L98
            61053 SEA FILE=MEDLINE ABB=ON PLU=ON ANTIOXID?
L100
            18939 SEA FILE=MEDLINE ABB=ON PLU=ON FLAV!NOID?/BI
L101
            32882 SEA FILE=MEDLINE ABB=ON PLU=ON FLAVONOIDS+NT/CT
L102
                    QUE ABB=ON PLU=ON TRANSITION ELEMENTS+NT/CT
L103
                 3 SEA FILE=MEDLINE ABB=ON PLU=ON L96 AND ((L100 OR L101 OR
L105
                    L102 OR L103))
                    SEL PLU=ON L39 1- CHEM:
L106
                                                          344 TERMS
            19788 SEA FILE=MEDLINE ABB=ON PLU=ON L106
L107
                 2 SEA FILE=MEDLINE ABB=ON PLU=ON L107 AND L96
L108
                    QUE ABB=ON PLU=ON FERRIOXAMIN? OR DEFEROXAMIN? OR DESF
L109
                    ERROXAMIN? OR DEFERRIOXAMIN?
L110
                    QUE ABB=ON PLU=ON EDETIC ACID/CT
                    OUE ABB=ON PLU=ON CP94
L111
                    QUE ABB=ON PLU=ON HYDROXAMIC ACIDS/CT
L112
                   OUE ABB=ON PLU=ON APOFERRITIN/CT
L113
                   OUE ABB=ON PLU=ON CDTA
L114
L115
                   OUE ABB=ON PLU=ON DTPA OR PENTATIC ACID
                   QUE ABB=ON PLU=ON PENICILLAMINE
L116
                   QUE ABB=ON PLU=ON BATHOCUPROINE
L117
                   QUE ABB=ON PLU=ON BATHOCUPROIN
L118
L119
               6 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND (L109 OR L110 OR L111
                   OR L112 OR L113 OR L114 OR L115 OR L116 OR L117 OR L118)
                1 SEA FILE=MEDLINE ABB=ON PLU=ON L119 AND L107
L120
               13 SEA FILE=MEDLINE ABB=ON PLU=ON L82 OR L87
L121
               10 SEA FILE=MEDLINE ABB=ON PLU=ON L96 OR L98 OR L105 OR L108 OR
L122
                   L119 OR L120
                 3 SEA FILE=MEDLINE ABB=ON PLU=ON L121 AND L122
Б179
```

=> s L82 or L87 or L179

L209 13 L82 OR L87 OR L179

=> file embase

FILE 'EMBASE' ENTERED AT 15:22:20 ON 17 JUL 2006 Copyright (c) 2006 Elsevier B.V. All rights reserved.

FILE COVERS 1974 TO 17 Jul 2006 (20060717/ED)

EMBASE has been reloaded. Enter HELP RLOAD for details.

EMBASE is now updated daily. SDI frequency remains weekly (default) and biweekly.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> d que L127

L124	7035	SEA	FILE=EMBASE	ABB=ON	PLU=ON	WANG S?/AU
L125						SHACKLEFORD R?/AU
L126						SHACKELFORD R?/AU
L127	4	SEA	FILE=EMBASE	ABB=ON	PLU=ON	L124 AND (L125 OR L126)

=> d que L135

L124	7035	SEA FILE=EMBASE ABB=ON	PLU=ON	WANG S?/AU
L125	5	SEA FILE=EMBASE ABB=ON	PLU=ON	SHACKLEFORD R?/AU
L126	27	SEA FILE=EMBASE ABB=ON	PLU=ON	SHACKELFORD R?/AU
L128	2332	SEA FILE=EMBASE ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA+UF/CT
L129	3044	SEA FILE=EMBASE ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA
L130	62	SEA FILE=EMBASE ABB=ON	PLU=ON	LOUIS BAR
L131	2	SEA FILE=EMBASE ABB=ON	PLU=ON	
L132	0	SEA FILE=EMBASE ABB=ON	PLU=ON	TELANGIECTASIA CEREBELLOOCULOCU
		TANEA		
L133	0	SEA FILE=EMBASE ABB=ON	PLU=ON	TELANGIECTASIA CEREBELLO
		OCULOCUTANEA		
L134	3053	SEA FILE=EMBASE ABB=ON	PLU=ON	(L128 OR L129 OR L130 OR L131
		OR L132 OR L133)		
L135	11	SEA FILE=EMBASE ABB=ON	PLU=ON	(L124 OR L125 OR L126) AND
		L134		

=> d que L178

```
1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
L1
                       1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN

2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN

6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L2
L3
L4
L5
L6
L7
                            OR L6)
                       1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT)"/CN
L8
L9
L10
                       1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN
L11
                      O SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
L12
L13
                            OR "DEFEROXAMINE METHANESULFONATE"/CN)
                      0 SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
L14
L15
L16
                       1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
L17
                       1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
L18
L19
                       1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
L20
```

```
6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
L21
             4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
L22
               C?/CN
            23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L23
               OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
               L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
             1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
L29
            24 SEA FILE=REGISTRY ABB=ON
                                        PLU=ON L23 OR L29
L31
L37
            22 SEA FILE=REGISTRY ABB=ON PLU=ON
                                                (117-39-5/BI OR 146426-40-6/B
               I OR 153-18-4/BI OR 17912-87-7/BI OR 446-72-0/BI OR 480-16-0/BI
                OR 480-18-2/BI OR 480-40-0/BI OR 480-41-1/BI OR 482-39-3/BI
               OR 490-46-0/BI OR 491-70-3/BI OR 491-80-5/BI OR 50-78-2/BI OR
               520-26-3/BI OR 520-27-4/BI OR 520-33-2/BI OR 520-36-5/BI OR
               522-12-3/BI OR 525-82-6/BI OR 577-85-5/BI OR 989-51-5/BI)
L38
             1 SEA FILE=REGISTRY ABB=ON PLU=ON 50-78-2
L39
            21 SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38
          7035 SEA FILE=EMBASE ABB=ON PLU=ON WANG S?/AU
L124
            5 SEA FILE=EMBASE ABB=ON PLU=ON SHACKLEFORD R?/AU
L125
            27 SEA FILE=EMBASE ABB=ON PLU=ON SHACKELFORD R?/AU
L126
             4 SEA FILE=EMBASE ABB=ON PLU=ON L124 AND (L125 OR L126)
L127
          2332 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA+UF/CT
L128
          3044 SEA FILE=EMBASE ABB=ON
                                      PLU=ON ATAXIA TELANGIECTASIA
L129
            62 SEA FILE=EMBASE ABB=ON PLU=ON LOUIS BAR
L130
             2 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTATICA
L131
             O SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLOOCULOCU
L132
               TANEA
             O SEA FILE-EMBASE ABB-ON PLU-ON TELANGIECTASIA CEREBELLO
L133
               OCULOCUTANEA
          3053 SEA FILE=EMBASE ABB=ON PLU=ON (L128 OR L129 OR L130 OR L131
L134
               OR L132 OR L133)
            11 SEA FILE=EMBASE ABB=ON PLU=ON (L124 OR L125 OR L126) AND
L135
               L134
         98621 SEA FILE=EMBASE ABB=ON PLU=ON CHELATING AGENT+NT/CT
L136
               SEL PLU=ON L31 1- CHEM:
                                             255 TERMS
L137
         61283 SEA FILE=EMBASE ABB=ON PLU=ON L137
L138
            14 SEA FILE=EMBASE ABB=ON PLU=ON L134 AND (L136 OR L138)
L139
         25033 SEA FILE=EMBASE ABB=ON PLU=ON FLAVONOID+NT/CT
L140
L141
         35447 SEA FILE=EMBASE ABB=ON PLU=ON ANTIOXIDANT+NT/CT
L142
             4 SEA FILE=EMBASE ABB=ON PLU=ON L139 AND (L140 OR L141)
                                             344 TERMS
L143
               SEL PLU=ON L39 1- CHEM:
         24101 SEA FILE=EMBASE ABB=ON PLU=ON L143
L144
             2 SEA FILE=EMBASE ABB=ON PLU=ON L139 AND L144
L145
            14 SEA FILE=EMBASE ABB=ON PLU=ON L139 OR L142 OR L145
L146
L147
            11 SEA FILE=EMBASE ABB=ON PLU=ON L127 OR L135
         31221 SEA FILE=EMBASE ABB=ON PLU=ON CHELAT?
L149
             4 SEA FILE=EMBASE ABB=ON PLU=ON L149 AND L134
L150
            14 SEA FILE=EMBASE ABB=ON PLU=ON L146 OR L150
L177
             4 SEA FILE=EMBASE ABB=ON PLU=ON L147 AND L177
L178
```

=> s L127 or L135 or L178

L210 11 L127 OR L135 OR L178

=> file biosis

See 11 12 76 2 ..

FILE 'BIOSIS' ENTERED AT 15:22:24 ON 17 JUL 2006 Copyright (c) 2006 The Thomson Corporation

FILE COVERS 1969 TO DATE.

CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 12 July 2006 (20060712/ED)

L29

L31

L37

```
=> d que L154
         10521 SEA FILE=BIOSIS ABB=ON PLU=ON WANG S?/AU
            52 SEA FILE=BIOSIS ABB=ON PLU=ON SHACKELFORD R?/AU
L152
             7 SEA FILE=BIOSIS ABB=ON PLU=ON SHACKLEFORD R?/AU
L153
             6 SEA FILE=BIOSIS ABB=ON PLU=ON L151 AND (L152 OR L153)
L154
=> d que L156
         10521 SEA FILE=BIOSIS ABB=ON PLU=ON WANG S?/AU
L151
            52 SEA FILE=BIOSIS ABB=ON PLU=ON SHACKELFORD R?/AU
L152
             7 SEA FILE=BIOSIS ABB=ON PLU=ON SHACKLEFORD R?/AU
L153
          3180 SEA FILE=BIOSIS ABB=ON PLU=ON ATAXIA TELANGIECTASIA
L155
            18 SEA FILE=BIOSIS ABB=ON PLU=ON (L151 OR L152 OR L153) AND
L156
                T-155
=> d que L176
              1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
L1
              1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
L_2
              1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
1.3
              1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
T.4
              1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
L5
              2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
L6
              6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L7
               OR L6)
             1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
L8
             1 SEA FILE=REGISTRY ABB=ON PLU=ON
                                                 EDTA/CN
L9
             1 SEA FILE=REGISTRY ABB=ON PLU=ON
                                                 "EDTA (CHELATING AGENT)"/CN
L10
                                                 DEFEROXAMINE B MESYLATE/CN
             1 SEA FILE=REGISTRY ABB=ON PLU=ON
L11
             O SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
L12
             1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
L13
                OR "DEFEROXAMINE METHANESULFONATE"/CN)
              O SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
L14
             1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
L15
             1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
L16
             1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
L17
             1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
L18
             1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
L19
              1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
L20
              6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
L21
              4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
L22
                C?/CN
             23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L23
                OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
                L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
              1 SEA FILE=REGISTRY ABB=ON PLU=ON
```

22 SEA FILE=REGISTRY ABB=ON PLU=ON (117-39-5/BI OR 146426-40-6/B

I OR 153-18-4/BI OR 17912-87-7/BI OR 446-72-0/BI OR 480-16-0/BI OR 480-18-2/BI OR 480-40-0/BI OR 480-41-1/BI OR 482-39-3/BI OR 490-46-0/BI OR 491-70-3/BI OR 491-80-5/BI OR 50-78-2/BI OR

24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29

70-51-9

```
520-26-3/BI OR 520-27-4/BI OR 520-33-2/BI OR 520-36-5/BI OR
                  522-12-3/BI OR 525-82-6/BI OR 577-85-5/BI OR 989-51-5/BI)
                1 SEA FILE=REGISTRY ABB=ON PLU=ON 50-78-2
L38
              21 SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38
L39
            10521 SEA FILE=BIOSIS ABB=ON PLU=ON WANG S?/AU
L151
           52 SEA FILE=BIOSIS ABB=ON PLU=ON SHACKELFORD R?/AU
L152
               7 SEA FILE=BIOSIS ABB=ON PLU=ON SHACKLEFORD R?/AU
L153
              6 SEA FILE=BIOSIS ABB=ON PLU=ON L151 AND (L152 OR L153)
L154
L155
L156
            3180 SEA FILE=BIOSIS ABB=ON PLU=ON ATAXIA TELANGIECTASIA
               18 SEA FILE=BIOSIS ABB=ON PLU=ON (L151 OR L152 OR L153) AND
                  L155
L157
            38028 SEA FILE=BIOSIS ABB=ON PLU=ON CHELAT?
                  SEL PLU=ON L31 1- CHEM : 255 TERMS
L158
L159
L160
59051 SEA FILE=BIOSIS ABB=ON PLU=ON L158
          31206 SEA FILE=BIOSIS ABB=ON PLU=ON L170

3 SEA FILE=BIOSIS ABB=ON PLU=ON L171 AND L168

0 SEA FILE=BIOSIS ABB=ON PLU=ON L168 AND (L161 OR L162)

18 SEA FILE=BIOSIS ABB=ON PLU=ON L154 OR L156

7 SEA FILE=BIOSIS ABB=ON PLU=ON L168 OR L172 OR L173

4 SEA FILE=BIOSIS ABB=ON PLU=ON L174 AND L175
L172
L173
L174
L175
L176
```

=> s L154 or L156 or L176

L211 18 L154 OR L156 OR L176

=> file uspatfull

FILE 'USPATFULL' ENTERED AT 15:22:28 ON 17 JUL 2006
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FILE COVERS 1971 TO PATENT PUBLICATION DATE: 13 Jul 2006 (20060713/PD)
FILE LAST UPDATED: 13 Jul 2006 (20060713/ED)
HIGHEST GRANTED PATENT NUMBER: US7076805
HIGHEST APPLICATION PUBLICATION NUMBER: US2006156447
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REVISED CLASS FIELDS (/NCL) LAST RELOADED: Feb 2006
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Feb 2006

=> d que L205

L202	1992	SEA	FILE=USPATFULL A	BB=ON	PLU=ON	WANG S?/AU
L203	5	SEA	FILE=USPATFULL A	BB=ON	PLU=ON	SHACKELFORD R?/AU
L204	3	SEA	FILE=USPATFULL A	BB=ON	PLU=ON	SHACKLEFORD R?/AU
L205	1	SEA	FILE=USPATFULL A	BB=ON	PLU=ON	L202 AND (L203 OR L204)

=> d que L206

```
1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
L1
             1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
L2
             1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
L3
             1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
L4
            1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
L5
            2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
L6
            6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L7
               OR L6)
             1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
L8
             1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
L9
             1 SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT)"/CN
L10
            1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN
L11
            O SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
L12
            1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
L13
               OR "DEFEROXAMINE METHANESULFONATE"/CN)
            O SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
L14
             1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
L15
            1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
L16
            1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
L17
            1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
L18
            1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
L19
            1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
L20
             6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
L21
            4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
L22
               C?/CN
            23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
               OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
               L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
             1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
L29
            24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
L31
            22 SEA FILE=REGISTRY ABB=ON PLU=ON (117-39-5/BI OR 146426-40-6/B
L37
               I OR 153-18-4/BI OR 17912-87-7/BI OR 446-72-0/BI OR 480-16-0/BI
                OR 480-18-2/BI OR 480-40-0/BI OR 480-41-1/BI OR 482-39-3/BI
               OR 490-46-0/BI OR 491-70-3/BI OR 491-80-5/BI OR 50-78-2/BI OR
               520-26-3/BI OR 520-27-4/BI OR 520-33-2/BI OR 520-36-5/BI OR
               522-12-3/BI OR 525-82-6/BI OR 577-85-5/BI OR 989-51-5/BI)
             1 SEA FILE=REGISTRY ABB=ON PLU=ON 50-78-2
L38
            21 SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38
L39
          5870 SEA FILE=USPATFULL ABB=ON PLU=ON L31
L183
          2355 SEA FILE=USPATFULL ABB=ON PLU=ON ATAXIA TELANGIECTASIA
L184
             6 SEA FILE-USPATFULL ABB=ON PLU=ON L183 AND L184
L185
          1795 SEA FILE=USPATFULL ABB=ON PLU=ON L39
L186
             1 SEA FILE-USPATFULL ABB-ON PLU-ON L185 AND L186
L187
          1992 SEA FILE=USPATFULL ABB=ON PLU=ON WANG S?/AU
L202
            5 SEA FILE=USPATFULL ABB=ON PLU=ON SHACKELFORD R?/AU
L203
             3 SEA FILE=USPATFULL ABB=ON PLU=ON SHACKLEFORD R?/AU
L204
             1 SEA FILE=USPATFULL ABB=ON PLU=ON (L202 OR L203 OR L204) AND
L206
               (L185 OR L187)
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=> s L205 or L206
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L212 1 L205 OR L206

=> file wpix

FILE 'WPIX' ENTERED AT 15:22:31 ON 17 JUL 2006 COPYRIGHT (C) 2006 THE THOMSON CORPORATION

33161534

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14 JUL 2006
                                             <20060714/UP>
FILE LAST UPDATED:
MOST RECENT DERWENT UPDATE:
                               200645
                                             <200645/DW>
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE
>>> FOR A COPY OF THE DERWENT WORLD PATENTS INDEX STN USER GUIDE,
    PLEASE VISIT:
 http://www.stn-international.de/training_center/patents/stn_guide.pdf <
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http://scientific.thomson.com/support/patents/coverage/latestupdates/
>>> PLEASE BE AWARE OF THE NEW IPC REFORM IN 2006, SEE
http://www.stn-international.de/stndatabases/details/ipc reform.html and
http://scientific.thomson.com/media/scpdf/ipcrdwpi.pdf <<<
>>> FOR FURTHER DETAILS ON THE FORTHCOMING DERWENT WORLD PATENTS
    INDEX ENHANCEMENTS PLEASE VISIT:
http://www.stn-international.de/stndatabases/details/dwpi_r.html <<<
'BIX' IS DEFAULT SEARCH FIELD FOR 'WPIX' FILE
=> d que L192
           5402 SEA FILE=WPIX ABB=ON PLU=ON
                                             WANG S?/AU
T-189
              O SEA FILE=WPIX ABB=ON
                                     PLU=ON
                                             SHACKLEFORD R?/AU
L190
              3 SEA FILE=WPIX ABB=ON PLU=ON
L191
                                             SHACKELFORD R?/AU
              1 SEA FILE-WPIX ABB-ON PLU-ON L189 AND (L190 OR L191)
L192
=> d que L196
           247 SEA FILE=WPIX ABB=ON PLU=ON ATAXIA TELANGIECTASIA/BIX
L188
           5402 SEA FILE=WPIX ABB=ON PLU=ON WANG S?/AU
L189
             O SEA FILE=WPIX ABB=ON PLU=ON
                                             SHACKLEFORD R?/AU
L190
              3 SEA FILE=WPIX ABB=ON PLU=ON SHACKELFORD R?/AU
L191
              O SEA FILE=WPIX ABB=ON PLU=ON L188 AND (L189 OR L190 OR L191)
L196
=> d que L207
           247 SEA FILE=WPIX ABB=ON PLU=ON ATAXIA TELANGIECTASIA/BIX
L188
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                                             WANG S?/AU
L189
L190
              O SEA FILE=WPIX ABB=ON PLU=ON
                                             SHACKLEFORD R?/AU
              3 SEA FILE=WPIX ABB=ON PLU=ON SHACKELFORD R?/AU
L191
                                             (RAAMBT/DCN OR RAGNQ8/DCN OR
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L195
                RAODFA/DCN OR RAOEMC/DCN OR RAOJBK/DCN OR RAOOTF/DCN OR
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                R01319/DCN OR R03811/DCN OR R03812/DCN OR R03949/DCN OR
                R04870/DCN OR R06069/DCN OR R06174/DCN OR R06413/DCN OR
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                R08504/DCN OR R09163/DCN OR R09222/DCN OR R09884/DCN OR
                R11605/DCN OR R19085/DCN OR R19452/DCN OR R20811/DCN OR
                R22037/DCN)
              2 SEA FILE=WPIX ABB=ON PLU=ON L188 AND L195
L197
              O SEA FILE=WPIX ABB=ON PLU=ON L197 AND ((L189 OR L190 OR
L207
                L191))
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L213 1 L192 OR L196 OR L207

=> dup rem L208 L209 L210 L211 L212 L213

FILE 'HCAPLUS' ENTERED AT 15:23:43 ON 17 JUL 2006

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L214 22 DUP REM L208 L209 L210 L211 L212 L213 (34 DUPLICATES REMOVED)

ANSWERS '1-12' FROM FILE HCAPLUS ANSWERS '13-15' FROM FILE MEDLINE ANSWERS '16-22' FROM FILE BIOSIS

=> => d ibib abs hitind hitstr L214 1-12; d iall L214 13-22 YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS, MEDLINE, BIOSIS' - CONTINUE? (Y)/N:y

L214 ANSWER 1 OF 22 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2

ACCESSION NUMBER:

2005:34588 HCAPLUS

DOCUMENT NUMBER:

142:127600

TITLE:

Methods and compositions using chelating

agents for treatment of ataxia-

telangiectasia and diseases associated with oxidative stress and genomic instability

INVENTOR(S): Wang, Suming; Shackelford, Rodney E.

PATENT ASSIGNEE(S):

USA

SOURCE:

U.S. Pat. Appl. Publ., 37 pp.

CODEN: USXXCO

DOCUMENT TYPE:

Patent

LANGUAGE:

English

FAMILY ACC. NUM. COUNT:

PATENT INFORMATION:

	PATENT NO.	KIND	DATE	AP	PLICATION NO.	DATE
						-
	US 2005009760	A1	20050113	US	2003-617943	20030711
	RITY APPLN. INFO.:				2003-617943	20030711
AB	This invention rela	tes to	the methods	and	pharmaceutical	compns. for
	treating diseases o	r disor	ders associa	ated	with oxidative	stress and/or

```
genomic instability. In particular, the invention relates to methods for
     treating ataxia telangiectasia (AT) and such disease
     states by administering a therapeutically effective amount of a
     chelating agent to increase genomic stability and/or decrease
     oxidative stress. The ferrous iron chelating agent
     desferrioxamine was used to increase the genomic stability of
     ataxia telangiectasia cells.
     ICM A61K031-7048
TC
     ICS A61K031-353; A61K031-198; A61K031-195
INCL 514027000; 514456000; 514566000
     1-10 (Pharmacology)
     Section cross-reference(s): 63
ST
     ataxia telangeictasia treatment chelating agent; oxidative
     stress disease treatment chelating agent; genomic instability disease treatment chelating agent; desferrioxamine treatment
     ataxia telangiectasia
     Alkenes, biological studies
IT
     RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);
     THU (Therapeutic use); BIOL (Biological study); USES (Uses)
        (alkatrienes, as chelating agent; chelating agents
        for treatment of ataxia telangiectasia and diseases
        associated with oxidative stress and genomic instability)
IT
     Ferritins
     RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);
     THU (Therapeutic use); BIOL (Biological study); USES (Uses)
        (apoferritins, as chelating agent; chelating agents
        for treatment of ataxia telangiectasia and diseases
        associated with oxidative stress and genomic instability)
TT
     Flavonoids
     RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);
     THU (Therapeutic use); BIOL (Biological study); USES (Uses)
        (as antioxidant; chelating agents for treatment of
        ataxia telangiectasia and diseases associated with
        oxidative stress and genomic instability)
IT
    Hydroxamic acids
     RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);
     THU (Therapeutic use); BIOL (Biological study); USES (Uses)
        (as chelating agent, tri-, intermediates; chelating
        agents for treatment of ataxia telangiectasia and
        diseases associated with oxidative stress and genomic instability)
IT
     Disease, animal
        (associated with oxidative stress or genomic instability;
        chelating agents for treatment of ataxia
        telangiectasia and diseases associated with oxidative stress and
        genomic instability)
IT
    Nervous system, disease
        (ataxia telangiectasia; chelating agents
        for treatment of ataxia telangiectasia and diseases
        associated with oxidative stress and genomic instability)
IT
     Cell membrane
        (chelating agent capable of crossing; chelating
        agents for treatment of ataxia telangiectasia and
        diseases associated with oxidative stress and genomic instability)
IT
     Transition metals, biological studies
     RL: ADV (Adverse effect, including toxicity); BSU (Biological study,
     unclassified); BIOL (Biological study)
        (chelating agents binding; chelating agents for
        treatment of ataxia telangiectasia and diseases
        associated with oxidative stress and genomic instability)
IT
     Animals
```

```
Chelating agents
    Combination chemotherapy
    Drug delivery systems
    Human
    Oxidative stress, biological
        (chelating agents for treatment of ataxia
        telangiectasia and diseases associated with oxidative stress and
       genomic instability)
IT
    Antioxidants
        (further treatment with; chelating agents for treatment of
       ataxia telangiectasia and diseases associated with
       oxidative stress and genomic instability)
ΙT
    Gene, animal
    RL: ADV (Adverse effect, including toxicity); BSU (Biological study,
     unclassified); BIOL (Biological study)
        (instability; chelating agents for treatment of
        ataxia telangiectasia and diseases associated with
        oxidative stress and genomic instability)
     117-39-5, Quercetin 153-18-4, Rutin 446-72-0,
    Genistein 480-16-0, Morin 480-18-2, Taxifolin
     480-40-0, Chrysin 480-41-1, Naringenin 482-39-3
     , Afzelin 490-46-0, Epicatechin 491-70-3, Luteolin
     491-80-5, Biochanin A 520-26-3, Hesperidin
     520-27-4, Diosmin 520-33-2, Hesperetin 520-36-5
     , Apigenin 522-12-3, Quercitrin 525-82-6, Flavone
     577-85-5, Flavonol 989-51-5, Epigallocatechin gallate
     17912-87-7, Myricitrin 146426-40-6, Flavopiridol
     RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);
     THU (Therapeutic use); BIOL (Biological study); USES (Uses)
        (as antioxidant; chelating agents for treatment of
        ataxia telangiectasia and diseases associated with
        oxidative stress and genomic instability)
     52-67-5, Penicillamine 60-00-4, EDTA, biological studies
     67-43-6, Diethylenetriamine-N,N,N',N'',N''-pentaacetic acid
     70-51-9D, Desferrioxamine, compds. 138-14-7, Desferal
     138-14-7D, Desferrioxamine B mesylate, compds. 13291-61-7
     , trans-1,2-Diaminocyclohexane-N,N,N',N'-tetraacetic acid
     14836-73-8, Ferrioxamine 73348-75-1 115900-75-9
     . CP94
     RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);
     THU (Therapeutic use); BIOL (Biological study); USES (Uses)
        (as chelating agent; chelating agents for treatment
        of ataxia telangiectasia and diseases associated with
        oxidative stress and genomic instability)
     7439-89-6, Iron, biological studies 7440-50-8, Copper, biological
IT
     studies
     RL: ADV (Adverse effect, including toxicity); BSU (Biological study,
     unclassified); BIOL (Biological study)
        (chelating agent of; chelating agents for treatment
        of ataxia telangiectasia and diseases associated with
        oxidative stress and genomic instability)
     75-91-2, tert-Butyl hydroperoxide 7447-39-4, Copper chloride (CuCl2),
     biological studies 7758-94-3, Ferrous chloride
     RL: ADV (Adverse effect, including toxicity); BSU (Biological study,
     unclassified); BIOL (Biological study)
        (chelating agents for treatment of ataxia
        telangiectasia and diseases associated with oxidative stress and
        genomic instability)
     50-78-2, Aspirin 70-51-9, Desferrioxamine
     RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);
```

THU (Therapeutic use); BIOL (Biological study); USES (Uses) (chelating agents for treatment of ataxia telangiectasia and diseases associated with oxidative stress and genomic instability) IT 117-39-5, Quercetin 153-18-4, Rutin 446-72-0, Genistein 480-16-0, Morin 480-18-2, Taxifolin 480-40-0, Chrysin 480-41-1, Naringenin 482-39-3 , Afzelin 490-46-0, Epicatechin 491-70-3, Luteolin 491-80-5, Biochanin A 520-26-3, Hesperidin 520-27-4, Diosmin 520-33-2, Hesperetin 520-36-5 , Apigenin 522-12-3, Quercitrin 525-82-6, Flavone 577-85-5, Flavonol 989-51-5, Epigallocatechin gallate 17912-87-7, Myricitrin 146426-40-6, Flavopiridol RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (as antioxidant; chelating agents for treatment of ataxia telangiectasia and diseases associated with oxidative stress and genomic instability) RN117-39-5 HCAPLUS 4H-1-Benzopyran-4-one, 2-(3,4-dihydroxyphenyl)-3,5,7-trihydroxy- (9CI) CN (CA INDEX NAME)

RN 153-18-4 HCAPLUS
CN 4H-1-Benzopyran-4-one, 3-[[6-O-(6-deoxy-α-L-mannopyranosyl)-β-Dglucopyranosyl]oxy]-2-(3,4-dihydroxyphenyl)-5,7-dihydroxy- (9CI) (CA
INDEX NAME)

Absolute stereochemistry. Rotation (+).

RN 446-72-0 HCAPLUS CN 4H-1-Benzopyran-4-one, 5,7-dihydroxy-3-(4-hydroxyphenyl)- (9CI) (CA INDEX NAME)

RN 480-16-0 HCAPLUS CN 4H-1-Benzopyran-4-one, 2-(2,4-dihydroxyphenyl)-3,5,7-trihydroxy- (9CI) (CA INDEX NAME)

RN 480-18-2 HCAPLUS CN 4H-1-Benzopyran-4-one, 2-(3,4-dihydroxyphenyl)-2,3-dihydro-3,5,7-trihydroxy-, (2R,3R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).

RN 480-40-0 HCAPLUS CN 4H-1-Benzopyran-4-one, 5,7-dihydroxy-2-phenyl- (9CI) (CA INDEX NAME)

RN 480-41-1 HCAPLUS CN 4H-1-Benzopyran-4-one, 2,3-dihydro-5,7-dihydroxy-2-(4-hydroxyphenyl)-, (2S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

RN 482-39-3 HCAPLUS CN 4H-1-Benzopyran-4-one, 3-[(6-deoxy- α -L-mannopyranosyl)oxy]-5,7-

dihydroxy-2-(4-hydroxyphenyl)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).

490-46-0 HCAPLUS RN

2H-1-Benzopyran-3,5,7-triol, 2-(3,4-dihydroxyphenyl)-3,4-dihydro-, CN (2R,3R) - (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).

491-70-3 HCAPLUS RN

4H-1-Benzopyran-4-one, 2-(3,4-dihydroxyphenyl)-5,7-dihydroxy- (9CI) (CA CN INDEX NAME)

491-80-5 HCAPLUS RN

4H-1-Benzopyran-4-one, 5,7-dihydroxy-3-(4-methoxyphenyl)- (9CI) (CA INDEX CNNAME)

RN 520-26-3 HCAPLUS

CN 4H-1-Benzopyran-4-one, 7-[[6-0-(6-deoxy-α-L-mannopyranosyl)-β-D-glucopyranosyl]oxy]-2,3-dihydro-5-hydroxy-2-(3-hydroxy-4-methoxyphenyl)-, (2S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

RN 520-27-4 HCAPLUS CN 4H-1-Benzopyran-4-one, 7-[[6-O-(6-deoxy- α -L-mannopyranosyl)- β -D-

glucopyranosyl]oxy]-5-hydroxy-2-(3-hydroxy-4-methoxyphenyl)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

RN 520-33-2 HCAPLUS

CN 4H-1-Benzopyran-4-one, 2,3-dihydro-5,7-dihydroxy-2-(3-hydroxy-4-methoxyphenyl)-, (2S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

RN 520-36-5 HCAPLUS

CN 4H-1-Benzopyran-4-one, 5,7-dihydroxy-2-(4-hydroxyphenyl)- (9CI) (CA INDEX NAME)

RN 522-12-3 HCAPLUS

CN 4H-1-Benzopyran-4-one, 3-[(6-deoxy- α -L-mannopyranosyl)oxy]-2-(3,4-dihydroxyphenyl)-5,7-dihydroxy- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

RN 525-82-6 HCAPLUS

CN 4H-1-Benzopyran-4-one, 2-phenyl- (9CI) (CA INDEX NAME)

RN 577-85-5 HCAPLUS

CN 4H-1-Benzopyran-4-one, 3-hydroxy-2-phenyl- (9CI) (CA INDEX NAME)

RN 989-51-5 HCAPLUS

CN Benzoic acid, 3,4,5-trihydroxy-, (2R,3R)-3,4-dihydro-5,7-dihydroxy-2-(3,4,5-trihydroxyphenyl)-2H-1-benzopyran-3-yl ester (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).

RN 17912-87-7 HCAPLUS

CN 4H-1-Benzopyran-4-one, 3-[(6-deoxy-α-L-mannopyranosyl)oxy]-5,7-dihydroxy-2-(3,4,5-trihydroxyphenyl)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).

RN 146426-40-6 HCAPLUS

CN 4H-1-Benzopyran-4-one, 2-(2-chlorophenyl)-5,7-dihydroxy-8-[(3S,4R)-3-hydroxy-1-methyl-4-piperidinyl]- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).

IT 52-67-5, Penicillamine 60-00-4, EDTA, biological studies 67-43-6, Diethylenetriamine-N,N,N',N'',N''-pentaacetic acid 70-51-9D, Desferrioxamine, compds. 138-14-7, Desferal 138-14-7D, Desferrioxamine B mesylate, compds. 13291-61-7 , trans-1,2-Diaminocyclohexane-N,N,N',N'-tetraacetic acid 14836-73-8, Ferrioxamine 73348-75-1 115900-75-9 RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (as chelating agent; chelating agents for treatment of ataxia telangiectasia and diseases associated with oxidative stress and genomic instability) 52-67-5 HCAPLUS RND-Valine, 3-mercapto- (9CI) (CA INDEX NAME) CN

Absolute stereochemistry.

RN 60-00-4 HCAPLUS CN Glycine, N,N'-1,2-ethanediylbis[N-(carboxymethyl)- (9CI) (CA INDEX NAME)

$$\begin{array}{c|c} \text{CH$_2$--CO$_2$H} & \text{CH$_2$--CO$_2$H} \\ | & | & | \\ \text{HO$_2$C$--CH$_2$--N$--CH$_2$--CO$_2$H} \end{array}$$

RN 67-43-6 HCAPLUS
CN Glycine, N,N-bis[2-[bis(carboxymethyl)amino]ethyl]- (7CI, 8CI, 9CI) (CALL INDEX NAME)

RN 70-51-9 HCAPLUS

CN Butanediamide, N'-[5-[[4-[[5-(acetylhydroxyamino)pentyl]amino]-1,4-dioxobutyl]hydroxyamino]pentyl]-N-(5-aminopentyl)-N-hydroxy-(9CI) (CA INDEX NAME)

PAGE 1-B

$$-$$
 (CH₂)₅-NH₂

RN 138-14-7 HCAPLUS

CN Butanediamide, N'-[5-[[4-[[5-(acetylhydroxyamino)pentyl]amino]-1,4-dioxobutyl]hydroxyamino]pentyl]-N-(5-aminopentyl)-N-hydroxy-, monomethanesulfonate (salt) (9CI) (CA INDEX NAME)

CM 1

CRN 75-75-2 CMF C H4 O3 S

CM 2

CRN 70-51-9

CMF C25 H48 N6 O8

PAGE 1-B

-(CH₂)₅-NH₂

RN 138-14-7 HCAPLUS

CN Butanediamide, N'-[5-[[4-[[5-(acetylhydroxyamino)pentyl]amino]-1,4-dioxobutyl]hydroxyamino]pentyl]-N-(5-aminopentyl)-N-hydroxy-, monomethanesulfonate (salt) (9CI) (CA INDEX NAME)

CM 1

CRN 75-75-2 CMF C H4 O3 S

CM 2

CRN 70-51-9 CMF C25 H48 N6 O8

PAGE 1-B

-(CH₂)₅-NH₂

RN 13291-61-7 HCAPLUS

CN Glycine, N,N'-(1R,2R)-1,2-cyclohexanediylbis[N-(carboxymethyl)-, rel-(9CI) (CA INDEX NAME)

Relative stereochemistry.

RN 14836-73-8 HCAPLUS
CN Iron, [N'-[5-[[4-[[5-[(acetyl-κΟ) (hydroxy-κΟ) amino]pentyl]amino]-1-(oxo-κΟ)-4-oxobutyl] (hydroxy-κΟ) amino]pentyl]-N-(5-aminopentyl)-N-(hydroxy-κΟ) butanediamidato(3-)-κΟ1]-, (OC-6-64)- (9CI) (CA INDEX NAME)

RN 73348-75-1 HCAPLUS CN 1,10-Phenanthroline, 2,9-dimethyl-4,7-diphenyl-, disulfo deriv. (9CI) (CA INDEX NAME)

RN 115900-75-9 HCAPLUS

CN 4(1H)-Pyridinone, 1,2-diethyl-3-hydroxy- (9CI) (CA INDEX NAME)

IT 70-51-9, Desferrioxamine

RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);

THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(chelating agents for treatment of ataxia

telangiectasia and diseases associated with oxidative stress and

genomic instability)

RN 70-51-9 HCAPLUS

CN Butanediamide, N'-[5-[[4-[[5-(acetylhydroxyamino)pentyl]amino]-1,4-

dioxobutyl]hydroxyamino]pentyl]-N-(5-aminopentyl)-N-hydroxy- (9CI) (CA INDEX NAME)

PAGE 1-B

-- (CH₂)₅-NH₂

L214 ANSWER 2 OF 22 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 3

ACCESSION NUMBER:

2005:107610 HCAPLUS

DOCUMENT NUMBER:

142:422753

TITLE:

Pharmacological manipulation of ataxia-

telangiectasia kinase activity as a treatment

for Parkinson's disease

AUTHOR(S):

Shackelford, Rodney Edwin; Manuszak, Ryan P.; Heard, Steven C.; Link, Charles J.; Wang,

Sumina

CORPORATE SOURCE:

Department of Pathology, Lousiana State University at

Shreveport, Shreveport, LA, 711030-3932, USA

SOURCE:

Medical Hypotheses (2005), 64(4), 736-741

CODEN: MEHYDY; ISSN: 0306-9877

PUBLISHER:

Elsevier Ltd.

DOCUMENT TYPE:

Journal; General Review

LANGUAGE:

English

AB A review. Parkinson's disease (PD) is a major cause of morbidity and mortality among older individuals. Although the causes of Parkinson's disease are multifactorial, considerable evidence indicates that elevated

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labile iron in the substantia nigra pars compacta plays an important role
in producing oxyradicals which subsequently damage nigro-striatal neurons.
Based on this several researchers have suggested that blood-brain barrier
crossing iron chelators might have clin. efficacy in treating
PD. Work demonstrating that iron chelators protect
nigro-striatal neurons in the N-methyl-4-phenyl-1,2,3,6-tetrahydropyridine
and 6-hydroxydopamine-induced rodent PD models supports this hypothesis.
Recently, we found that the ATM gene product (mutated in ataxia-
telangiectasia, A-T), is required for cell survival and genomic
stability maintenance following exposure to low labile iron concns. Iron
chelators (desferal, quercetin, and apoferritin) also increase A-T
cell genomic stability and viability, and activate ATM-dependent cellular
events in normal cells. Addnl. Atm-deficient mice exhibit a selective
loss of dopaminergic nigro-striatal neurons. Based on this, we propose
that iron chelators protect the substantia nigra pars compacta
not only by chelating labile iron and reducing oxyradical
formation, but also by inducing ATM activity, leading to increased
oxidative stress resistance and DNA repair. Support for this hypothesis
comes from the recent observation that the iron chelating
flavonoid quercetin both directly activates ATM and protects neuronal
cells from the toxic effects of the N-methyl-4-phenyl-1,2,3,6-
tetrahydropyridine. Therefore since; (1) ATM is required for iron toxicity resistance, (2) iron chelators such as quercetin,
desferal, and apoferritin induce ATM activity and/or ATM-dependent events,
and (3), Atm-deficient mice preferentially lose dopaminergic
nigro-striatal neurons, we propose that ATM activity has an important
function in PD. Furthermore, pharmacol. manipulation of ATM activity via
iron chelation might have clin. efficacy in PD treatment.
1-0 (Pharmacology)
review ataxia telangiectasia kinase iron
chelation Parkinson disease antiparkinsonian; desferal quercetin
apoferritin Parkinson disease antiparkinsonian review
Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
   (ATM (ataxia telangiectasia mutated); pharmacol.
   manipulation of mutated ataxia telangiectasia
   kinase activity via iron chelation showed efficacy in rodent
```

PD models and may increase SNpc neuronal cell survival and slow clin. progression of Parkinson's disease)

Ferritins IT

CC

IT

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(apoferritins; pharmacol. manipulation of mutated ataxia telangiectasia kinase activity via iron chelator apoferritin may increase SNpc neuronal cell survival and slow clin. progression of Parkinson's disease)

IT Antiparkinsonian agents Brain

Chelation

Parkinson's disease

(pharmacol. manipulation of mutated ataxia telangiectasia kinase activity via iron chelation showed efficacy in rodent PD models and may increase SNpc neuronal cell survival and slow clin. progression of Parkinson's disease)

IT Brain

(substantia nigra, pars compacta; pharmacol. manipulation of mutated ataxia telangiectasia kinase activity via iron chelation may increase neuronal cell survival in substantia nigra pars compacta and show clin. efficacy in Parkinson's disease)

7439-89-6, Iron, biological studies IT

- 32 -

RL: BSU (Biological study, unclassified); BIOL (Biological study) (pharmacol. manipulation of mutated ataxia telangiectasia kinase activity via iron chelation showed efficacy in rodent PD models and may increase SNpc neuronal cell survival and slow clin. progression of Parkinson's disease) IT 138-14-7, Desferal RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (pharmacol. manipulation of mutated ataxia telangiectasia kinase activity via iron chelator desferal may increase SNpc neuronal cell survival and slow clin. progression of Parkinson's disease) IT 117-39-5, Quercetin RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (pharmacol. manipulation of mutated ataxia telangiectasia kinase activity via iron chelator quercetin may increase SNpc neuronal cell survival and slow clin. progression of Parkinson's disease) IT **138-14-7**, Desferal RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (pharmacol. manipulation of mutated ataxia telangiectasia kinase activity via iron chelator desferal may increase SNpc neuronal cell survival and slow clin. progression of Parkinson's disease) 138-14-7 HCAPLUS RNCN Butanediamide, N'-[5-[[4-[[5-(acetylhydroxyamino)pentyl]amino]-1,4dioxobutyl]hydroxyamino]pentyl]-N-(5-aminopentyl)-N-hydroxy-, monomethanesulfonate (salt) (9CI) (CA INDEX NAME) CM 1 CRN 75~75-2 CMF C H4 O3 S CM 2 CRN 70-51-9 CMF C25 H48 N6 O8

PAGE 1-B

- (CH₂)₅-NH₂

117-39-5, Quercetin TT

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(pharmacol. manipulation of mutated ataxia

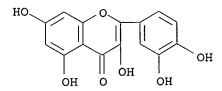
telangiectasia kinase activity via iron chelator

quercetin may increase SNpc neuronal cell survival and slow clin.

progression of Parkinson's disease)

117-39-5 HCAPLUS RN

4H-1-Benzopyran-4-one, 2-(3,4-dihydroxyphenyl)-3,5,7-trihydroxy- (9CI) CN (CA INDEX NAME)



REFERENCE COUNT:

THERE ARE 74 CITED REFERENCES AVAILABLE FOR THIS 74 RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 3 OF 22 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 4

2005:465449 HCAPLUS ACCESSION NUMBER:

143:277995 DOCUMENT NUMBER:

Pharmacologic manipulation of the ataxia-TITLE:

telangiectasia mutated gene product as an

intervention in age-related disease

Shackelford, Rodney E. AUTHOR (S):

Department of Pathology, Lousiana State University at CORPORATE SOURCE:

Shreveport, Shreveport, LA, 711030-3932, USA

Medical Hypotheses (2005), 65(2), 363-369 SOURCE:

CODEN: MEHYDY; ISSN: 0306-9877

Elsevier Ltd. PUBLISHER:

Journal; General Review DOCUMENT TYPE:

LANGUAGE: English

A review. Ataxia-telangiectasia (A-T) is an autosomal recessive disorder characterized by progressive ataxia, elevated cancer incidence, and premature aging. A-T cells, Atm-deficient mice, and individuals with A-T show increased oxidant sensitivity, genomic instability, altered IGF-1 and p53 signaling, and rapid telomere shortening compared to normal controls. The gene mutated in A-T, ATM, regulates DNA repair, IGF-1 and p53 signaling, age pigment removal, antioxidant capacity, and telomere maintenance - pathways involved in and often attenuated with aging. Interestingly, flavonoids with chemopreventative effects, such as quercetin, genistein, and epigallocatechin gallate activate ATM. Since ATM activates pathways which increase genomic stability, oxidant resistance, and/or telomere stability, and since many diseases of old age (i.e., cancer, cardiovascular and neurodegenerative disease), result from attenuation of these pathways, pharmacol. manipulation of ATM activity via flavonoid intake may prove

useful in slowing the appearance of age-associated disease.

1-0 (Pharmacology) CC

review genetic mutation flavonoid aging ataxia ST

1413 C Sec. 5

telangiectasia

Nervous system, disease IT

(ataxia telangiectasia; pharmacol. manipulation of

ATM gene activity via flavonoid may be useful in slowing appearance of age-associated disease in human by activating pathways which increase

genomic stability, oxidant resistance and telomere stability)

REFERENCE COUNT:

11/15 - +1

73 THERE ARE 73 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 4 OF 22 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 5

ACCESSION NUMBER:

2004:710500 HCAPLUS

DOCUMENT NUMBER:

141:236524

TITLE:

Iron chelators increase the resistance of

Ataxia telangiectasia cells to

oxidative stress

AUTHOR (S):

Shackelford, Rodney E.; Manuszak, Ryan P.;

Johnson, Cybele D.; Hellrung, Daniel J.; Link, Charles

J.; Wang, Suming

CORPORATE SOURCE:

Iowa Cancer Research Foundation, Urbandale, IA, 50322,

USA

SOURCE:

DNA Repair (2004), 3(10), 1263-1272

CODEN: DRNEAR; ISSN: 1568-7864

PUBLISHER:

Elsevier B.V.

DOCUMENT TYPE:

Journal

LANGUAGE:

English

Ataxia telangiectasia (A-T) is an autosomal recessive disorder characterized by immune dysfunction, genomic instability, chronic oxidative damage, and increased cancer incidence. Previously, desferal was found to increase the resistance of A-T, but not normal cells to exogenous oxidative stress in the colony forming-efficiency assay, suggesting that iron metabolism is dysregulated in A-T. Since desferal both chelates iron and modulates gene expression, the authors tested the effects of apoferritin and the iron chelating flavonoid quercetin on A-T cell colony-forming ability. The authors demonstrate that apoferritin and quercetin increase the ability of A-T cells to form colonies. The authors also show that labile iron levels are significantly elevated in Atm-deficient mouse sera compared to syngeneic wild type mice. Our findings support a role for labile iron acting as a Fenton catalyst in A-T, contributing to the chronic oxidative stress seen in this disease. Our findings further suggest that iron chelators might promote the survival of A-T cells and hence, individuals with A-T.

1-12 (Pharmacology) CC

quercetin apoferritin iron chelator oxidative stress ataxia ST telangiectasia

Ferritins TΨ

> RL: BSU (Biological study, unclassified); BIOL (Biological study) (apoferritins; iron chelators increase resistance of Ataxia telangiectasia cells to oxidative stress)

Nervous system, disease TT

(ataxia telangiectasia; iron chelators increase resistance of Ataxia telangiectasia cells to oxidative stress)

Antioxidants IT

Chelating agents

Oxidative stress, biological

(iron chelators increase resistance of Ataxia telangiectasia cells to oxidative stress)

IT Reactive oxygen species RL: BSU (Biological students)

RL: BSU (Biological study, unclassified); BIOL (Biological study)

(iron chelators increase resistance of Ataxia

telangiectasia cells to oxidative stress)

IT 7439-89-6, Iron, biological studies 7782-44-7D, Oxygen, reactive species

RL: BSU (Biological study, unclassified); BIOL (Biological study)

(iron chelators increase resistance of Ataxia

telangiectasia cells to oxidative stress)

IT 117-39-5, Quercetin

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL

(Biological study); USES (Uses)

(iron chelators increase resistance of Ataxia

telangiectasia cells to oxidative stress)

IT 117-39-5, Quercetin

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL

(Biological study); USES (Uses)

(iron chelators increase resistance of Ataxia

telangiectasia cells to oxidative stress)

RN 117-39-5 HCAPLUS

CN 4H-1-Benzopyran-4-one, 2-(3,4-dihydroxyphenyl)-3,5,7-trihydroxy- (9CI)

(CA INDEX NAME)

REFERENCE COUNT:

THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 5 OF 22 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 6

ACCESSION NUMBER: 2003:985326 HCAPLUS

DOCUMENT NUMBER: 140:175941

TITLE: Functional expression of ATM gene carried by HSV

amplicon vector in vitro and in vivo

AUTHOR(S): Qi, J.; Shackelford, R.; Manuszak, R.; Cheng, D.; Smith, M.; Link, C. J.; Wang, S.

CORPORATE SOURCE: Human Gene Therapy Research Institute, Stoddard Cancer

Research Institute, IA, USA

SOURCE: Gene Therapy (2004), 11(1), 25-33

CODEN: GETHEC; ISSN: 0969-7128

PUBLISHER: Nature Publishing Group

DOCUMENT TYPE: Journal
LANGUAGE: English
AB Ataxia-telangiectasia (AT) is a human autosomal

recessive disease with a pleiotropic phenotype characterized by cerebellar degeneration, immunodeficiency, premature aging, cancer predisposition, and radiation sensitivity. The gene mutated in AT, ATM (for AT-mutated), had been cloned and found to have ionizing radiation and oxidative stress-inducible kinase activity. No treatment can stop the progression of the disease. In this study, the complete open-reading frame of ATM

of the disease. In this study, the complete open-reading frame of ATM cDNA was cloned into a Herpes simplex virus type-1 (HSV-1) amplicon vector (pTO-ATM), and the transduction of cultured AT cells was demonstrated by immunohistochem. and Western blot anal. Functional gene expression was evaluated by cell colony-forming assays following exposure to oxidative stress. The survival of AT cells with ATM gene transduction was about

100% higher compared to nontransduced cells after t-Bu hydroperoxide treatments. Next, the normal ATM gene expression in different regions of the rat brain was studied. Immunohistochem. staining demonstrated weak endogenous ATM protein expression in neurons of the caudate-putamen, with significantly higher levels of expression detected in neurons in other brain regions. Exogenous ATM gene expression from pTO-ATM after viral transduction in the caudate-putamen of the adult rat was examined At 3 days after injection of the pTO-ATM viral vector, abundant pos. ATM staining of the neurons was found at the injection sites, in comparison to the controls. These data demonstrate that the relatively large ATM cDNA can be transduced and expressed in vitro and in vivo from an HSV amplicon viral vector. These data provide initial evidence that the replacement of the ATM gene into the cells of AT patients might be possible some day. 3-2 (Biochemical Genetics)

CC

Section cross-reference(s): 10, 14

IT Proteins

ಾಡಚಿತ್ರ.

RL: BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)

(ATM (ataxia telangiectasia mutated); functional

expression of ATM cDNA carried by HSV amplicon vector in vitro and in rat brain)

Nervous system, disease

(ataxia telangiectasia; functional expression of ATM cDNA carried by HSV amplicon vector in vitro and in rat brain)

IT

RL: BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)

(for ATM (ataxia telangiectasia mutated);

functional expression of ATM cDNA carried by HSV amplicon vector in vitro and in rat brain)

REFERENCE COUNT:

THERE ARE 67 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

1-m- - . . .

L214 ANSWER 6 OF 22 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 7

ACCESSION NUMBER: 2003:705261 HCAPLUS

DOCUMENT NUMBER: 140:105085

TITLE: Desferrioxamine treatment increases the genomic

stability of Ataxia-telangiectasia

cells

AUTHOR (S): Shackelford, Rodney E.; Manuszak, Ryan P.;

Johnson, Cybele D.; Hellrung, Daniel J.; Steele,

Timothy A.; Link, Charles J.; Wang, Suming

CORPORATE SOURCE: Osteopathic Medical Center, Des Moines University, Des

Moines, IA, 50309, USA

DNA Repair (2003), 2(9), 971-981 SOURCE:

CODEN: DRNEAR; ISSN: 1568-7864

PUBLISHER: Elsevier Science B.V.

DOCUMENT TYPE: Journal LANGUAGE: English

Ataxia-telangiectasia (AT) is an autosomal recessive disorder characterized by genomic instability, chronic oxidative damage, and increased cancer incidence. Compared to normal cells, AT cells exhibit unusual sensitivity to exogenous oxidants, including t-Bu hydroperoxide (t-BOOH). Since ferritin releases labile iron under oxidative stress (which is chronic in AT) and labile iron mediates the toxic effects of t-Bu hydroperoxide, we hypothesized that chelation of intracellular labile iron would increase the genomic stability of AT cells, with and without exogenous oxidative stress. Here we report that desferrioxamine treatment increases the plating efficiency of AT, but not normal cells, in the colony forming-efficiency assay (a

method often used to measure genomic stability). Addnl., desferrioxamine increases AT, but not normal cell resistance, to t-Bu hydroperoxide in this assay. Last, AT cells exhibit increased sensitivity to the toxic effects of FeCl2 in the colony forming-efficiency assay and fail to demonstrate a FeCl2-induced G2 checkpoint response when compared to normal cells. Our data indicates that: (1) chelation of labile iron increases genomic stability in AT cells, but not normal cells; and (2) AT cells exhibit deficits in their responses to iron toxicity. While preliminary, our findings suggest that AT might be, in part, a disorder of iron metabolism and treatment of individuals with AT with desferrioxamine might have clin. efficacy.

CC 1-11 (Pharmacology)

Section cross-reference(s): 14

ST iron chelator desferrioxamine genomic stability Ataxia telangiectasia

IT Nervous system, disease

(ataxia telangiectasia; iron chelator

desferrioxamine increases genomic stability of Ataxia

telangiectasia cells: iron metabolism role in AT pathogenesis)

IT Cell cycle

(checkpoint, G2; iron chelator desferrioxamine increases genomic stability of Ataxia telangiectasia cells: iron metabolism role in AT pathogenesis)

IT Chelating agents

Human

Oxidative stress, biological

(iron chelator desferrioxamine increases genomic stability of Ataxia telangiectasia cells: iron metabolism role in AT pathogenesis)

IT 50-78-2, Aspirin 70-51-9, Desferrioxamine

RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(iron chelator desferrioxamine increases genomic stability of Ataxia telangiectasia cells: iron metabolism role in AT pathogenesis)

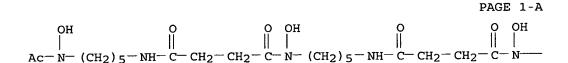
IT 70-51-9, Desferrioxamine

RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (iron chelator desferrioxamine increases genomic stability of

Ataxia telangiectasia cells: iron metabolism role in AT pathogenesis)

RN 70-51-9 HCAPLUS

CN Butanediamide, N'-[5-[[4-[[5-(acetylhydroxyamino)pentyl]amino]-1,4-dioxobutyl]hydroxyamino]pentyl]-N-(5-aminopentyl)-N-hydroxy- (9CI) (CA INDEX NAME)



PAGE 1-B

-- (CH₂)₅ -- NH₂

REFERENCE COUNT:

1. 1. 5 1. 7. 7. 5 E. C.

27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 7 OF 22 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 8

ACCESSION NUMBER:

2003:695693 HCAPLUS

DOCUMENT NUMBER:

140:124579

TITLE:

ATM-dependent and -independent gene expression changes in response to oxidative stress, gamma irradiation,

and UV irradiation

AUTHOR (S):

Heinloth, Alexandra N.; Shackelford, Rodney E. ; Innes, Cynthia L.; Bennett, Lee; Li, Leping; Amin, Rupesh P.; Sieber, Stella O.; Flores, Kristina G.;

Bushel, Pierre R.; Paules, Richard S.

CORPORATE SOURCE:

Growth Control and Cancer Group, National Institute of Environmental Health Sciences, Research Triangle Park,

NC, 27709, USA

SOURCE:

Radiation Research (2003), 160(3), 273-290

CODEN: RAREAE; ISSN: 0033-7587

PUBLISHER:

Radiation Research Society

DOCUMENT TYPE:

Journal English

LANGUAGE:

Ataxia telangiectasia (AT) is an autosomal recessive disorder characterized by progressive cerebellar degeneration, immunodeficiencies, telangiectasias, sensitivity to ionizing radiation, and high predisposition for malignancies. The ataxia telangiectasia mutated (ATM) gene encodes a protein (ATM) with serine/threonine kinase activity. DNA-double strand breaks are known to increase its kinase activity. While cells from individuals with AT are attenuated in their G1-, S- and G2-phase cell cycle checkpoint functions in response to γ irradiation and oxidative stress, their response to UV irradiation appears to be equivalent to that of wild-type cells. In this

we investigated changes in gene expression in response to γ irradiation, oxidative stress, and UV irradiation, focusing on the dependence on ATM. Doses for all three treatments were selected that resulted in roughly an equivalent induction of a G1 checkpoint response and inhibition of progression through S phase. To investigate gene expression changes, logarithmically growing wild-type and AT dermal diploid fibroblasts were exposed to either γ radiation (5 Gy), oxidative stress (75 μM t-butyl-hydroperoxide), or UV radiation (7.5 J/m2), and RNA was harvested 6 h after treatment. Gene expression anal. was performed using the NIEHS Human ToxChip 2.0 with approx. 1900 cDNA clones representing known genes and ESTs. All three treatments resulted in distinct patterns of gene expression changes, as shown previously. ATM-dependent and ATM-independent components were detected within these patterns, as were novel indications of involvement of ATM in regulation of transcription factors such as SP1, AP1 and MTF1.

8-7 (Radiation Biochemistry) CC

TΤ Nervous system, disease

(ataxia telangiectasia; ATM-dependent and

-independent gene expression changes in response to oxidative stress, γ -irradiation, and UV)

REFERENCE COUNT:

THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 8 OF 22 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 9

ACCESSION NUMBER: 2001:455746 HCAPLUS

DOCUMENT NUMBER: 135:193892

TITLE: The Ataxia telangiectasia gene

product is required for oxidative stress-induced G1

and G2 checkpoint function in human fibroblasts

AUTHOR(S): Shackelford, Rodney E.; Innes, Cynthia L.;

Sieber, Stella O.; Heinloth, Alexandra N.; Leadon,

Steven A.; Paules, Richard S.

CORPORATE SOURCE: Growth Control and Cancer Group, NIEHS, National

Institutes of Health, Research Triangle Park, NC,

27709, USA

SOURCE: Journal of Biological Chemistry (2001), 276(24),

21951-21959

CODEN: JBCHA3; ISSN: 0021-9258

PUBLISHER: American Society for Biochemistry and Molecular

Biology

DOCUMENT TYPE: Journal LANGUAGE: English

Ataxia telangiectasia (AT) is an autosomal recessive disorder characterized by neuronal degeneration accompanied by ataxia, telangiectasias, acute cancer pre-disposition, and sensitivity to ionizing radiation (IR). Cells from individuals with AT show unusual sensitivity to IR, severely attenuated cell cycle checkpoint functions, and poor p53 induction in response to IR compared with normal human fibroblasts (NHFs). The gene mutated in AT (ATM) has been cloned, and its product, pATM, has IR-inducible kinase activity. The AT phenotype has been suggested to be a consequence, at least in part, of an inability to respond appropriately to oxidative damage. To test this hypothesis, we examined the ability of NHFs and AT dermal fibroblasts to respond to t-Bu hydroperoxide and IR treatment. AT fibroblasts exhibit, in comparison to NHFs, increased sensitivity to the toxicity of t-Bu hydroperoxide, as measured by colony-forming efficiency assays. Unlike NHFs, AT fibroblasts fail to show G1 and G2 phase checkpoint functions or to induce p53 in response to t-Bu hydroperoxide. Treatment of NHFs with t-Bu hydroperoxide activates pATM-associated kinase activity. Our results indicate that pATM is involved in responding to certain aspects of oxidative damage and in signaling this information to downstream effectors of the cell cycle checkpoint functions. Our data further suggest that some of the pathologies seen in AT could arise as a consequence of an inability to respond normally to oxidative damage.

CC 14-10 (Mammalian Pathological Biochemistry)

ST Ataxia telangiectasia gene oxidative stress cell cycle fibroblast

IT Gene, animal

RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)

(AT; Ataxia telangiectasia gene product is required

for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)

IT Fibroblast

Oxidative stress, biological

Phenotypes

Signal transduction, biological

(Ataxia telangiectasia gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human

fibroblasts)

IT p53 (protein)

いっっつい

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(Ataxia telangiectasia gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)

IT Interphase (cell cycle)

> (G1-phase; Ataxia telangiectasia gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)

Interphase (cell cycle) IT

> (G2-phase; Ataxia telangiectasia gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)

IT Neoplasm

(acute cancer pre-disposition; Ataxia telangiectasia gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)

IT Nervous system

(ataxia telangiectasia; Ataxia

telangiectasia gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)

IT

(degeneration; Ataxia telangiectasia gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)

IT Ionizing radiation

(sensitivity to; Ataxia telangiectasia gene product is required for oxidative stress-induced G1 and G2 checkpoint function

IT 182970-53-2, gene ATM protein

in human fibroblasts)

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(Ataxia telangiectasia gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)

REFERENCE COUNT:

50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 9 OF 22 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 10

ACCESSION NUMBER:

2001:512887 HCAPLUS 135:267656

DOCUMENT NUMBER: TITLE:

Caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not

meiotic defects caused by loss of ataxia telangiectasia-mutated (Atm) gene function

Morita, Y.; Maravei, D. V.; Bergeron, L.; Wang, AUTHOR(S):

S.; Perez, G. I.; Tsutsumi, O.; Taketani, Y.;
Asano, M.; Horai, R.; Korsmeyer, S. J.; Iwakura, Y.;

Yuan, J.; Tilly, J. L.

CORPORATE SOURCE: Vincent Center for Reproductive Biology, Department of

Obstetrics and Gynecology, Massachusetts General Hospital/Harvard Medical School, Boston, MA, 02114,

USA

SOURCE: Cell Death and Differentiation (2001), 8(6), 614-620

CODEN: CDDIEK; ISSN: 1350-9047

PUBLISHER:

Nature Publishing Group

DOCUMENT TYPE:

Journal

LANGUAGE:

English

It is well established that programmed cell death claims up to two-thirds

of the oocytes produced during gametogenesis in the developing fetal ovaries. However, the mechanisms underlying prenatal germ cell loss in females remain poorly understood. Herein the authors report that caspase-11 null female mice are born with a reduced number of oocyte-containing primordial follicles. This phenotype is likely due to failed cytokine processing known to occur in caspase-11 mutants since neonatal female mice lacking both interleukin (IL)- 1α and IL- 1β also exhibit a reduced endowment of primordial follicles. In addition, germ cell death in wild-type fetal ovaries cultured ex vivo is suppressed by either cytokine, likely via ligand activation of type 1 IL-1 receptors expressed in fetal germ cells. Normal oocyte endowment can be restored in caspase-11 null female mice by simultaneous inactivation of the gene encoding the cell death executioner enzyme, caspase-2. However, caspase-2 deficiency cannot overcome gametogenic failure resulting from meiotic recombination defects in ataxia telangiectasia-mutated (Atm) null female Thus, genetically distinct mechanisms exist for developmental deletion of oocytes via programmed cell death, one of which probably functions as a meiotic quality-control checkpoint that cannot be overridden.

CC 2-10 (Mammalian Hormones)

Section cross-reference(s): 13

ST IL1 caspase ovary germ cell apoptosis ataxia

telangiectasia

IT Gene, animal

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(Bax; caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of ataxia telangiectasia-mutated gene function)

IT Nervous system

(ataxia telangiectasia; caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of ataxia telangiectasia-mutated gene function)

IT Apoptosis

Gamete and Germ cell

Meiosis

(caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of ataxia telangiectasia-mutated gene function)

IT Interleukin 1α

Interleukin 1ß

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of ataxia telangiectasia-mutated gene function)

IT Ovary

(follicle; caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of ataxia telangiectasia-mutated gene function)

IT Egg

(oocyte; caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of ataxia telangiectasia-mutated gene function)

IT Interleukin 1 receptors

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(type I; caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of ataxia telangiectasia-mutated gene function)

IT 182372-14-1, caspase 2 216503-96-7, caspase 11

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of ataxia telangiectasia-mutated gene function)

REFERENCE COUNT:

THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 10 OF 22 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 11

ACCESSION NUMBER:

1999:725087 HCAPLUS

DOCUMENT NUMBER:

132:62619

TITLE:

Lack of involvement of ataxia

telangiectasia mutated (ATM) in regulation of nuclear factor- κB (NF- κB) in human diploid

fibroblasts

AUTHOR (S):

Ashburner, Brian P.; Shackelford, Rodney E.; Baldwin, Albert S., Jr.; Paules, Richard S.

CORPORATE SOURCE:

Lineberger Comprehensive Cancer Center, University of North Carolina School of Medicine, Chapel Hill, NC,

27599, USA

SOURCE:

Cancer Research (1999), 59(21), 5456-5460

CODEN: CNREA8; ISSN: 0008-5472

PUBLISHER: AACR Subscription Office

DOCUMENT TYPE: LANGUAGE: Journal English

It has been suggested that the cellular response to exposure to ionizing AB radiation involves activation of the transcription factor nuclear factor- κB (NF- κB) and that this response is defective in cells from individuals with ataxia telangiectasia (AT). In one study, it was found that SV40 large T-transformed cells derived from a patient null for the AT mutated (ATM) gene exhibited constitutive activation of NF-κB and that in those cells, inhibition of NF- κB by expression of a modified form of $I\kappa B\alpha$ led to correction of the radiosensitivity associated with the AT phenotype. From those data, it was suggested that NF- κB played a role in the AT phenotype. We show here that normal diploid cells derived from AT patients do not exhibit constitutive activation of NF-kB. Furthermore, we provide data that the transformation process associated with SV40 large T antigen expression in AT-/- cells leads to aberrant cellular responses. Our studies highlight the importance of using diploid, nontransformed AT-/- cells for in vitro studies relevant to the AT phenotype whenever possible.

CC 14-14 (Mammalian Pathological Biochemistry)
 Section cross-reference(s): 3, 8

ST ataxia telangiectasia gene ATM NFkappaB fibroblast

IT Gene, animal

RL: ADV (Adverse effect, including toxicity); BIOL (Biological study) (ATM; lack of involvement of ataxia telangiectasia mutated (ATM) in regulation of NF-κB in human diploid fibroblasts in relation to radiosensitivity)

IT Phosphoproteins

RL: BAC (Biological activity or effector, except adverse); BSU (Biological

•

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study, unclassified); BIOL (Biological study)
        (IκB-\alpha (inhibitor of RNA formation factor NF-κB,
        a); lack of involvement of ataxia
        telangiectasia mutated (ATM) in regulation of NF-κB in
        human diploid fibroblasts in relation to radiosensitivity)
     Transcription factors
IT
     RL: BAC (Biological activity or effector, except adverse); BPR (Biological
     process); BSU (Biological study, unclassified); BIOL (Biological study);
     PROC (Process)
        (NF-\kappaB (nuclear factor \kappaB); lack of involvement of
        ataxia telangiectasia mutated (ATM) in regulation of
        NF-κB in human diploid fibroblasts in relation to
        radiosensitivity)
     Nervous system
IT
        (ataxia telangiectasia; lack of involvement of
        ataxia telangiectasia mutated (ATM) in regulation of
        NF-κB in human diploid fibroblasts in relation to
        radiosensitivity)
     Transformation, neoplastic (fibroblast; lack of involvement of ataxia
IT
        telangiectasia mutated (ATM) in regulation of NF-kB in
        human diploid fibroblasts in relation to radiosensitivity)
     Fibroblast
TΤ
     Ionizing radiation
        (lack of involvement of ataxia telangiectasia
        mutated (ATM) in regulation of NF-κB in human diploid fibroblasts
        in relation to radiosensitivity)
     Fibroblast
IT
        (transformation; lack of involvement of ataxia
        telangiectasia mutated (ATM) in regulation of NF-kB in
        human diploid fibroblasts in relation to radiosensitivity)
                                THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS
REFERENCE COUNT:
                         23
                                RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
L214 ANSWER 11 OF 22 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 12
                         1999:221513 HCAPLUS
ACCESSION NUMBER:
DOCUMENT NUMBER:
                         131:54809
                         Cell cycle control, checkpoint mechanisms, and
TITLE:
                         genotoxic stress
                         Shackelford, Rodney E.; Kaufmann, William
AUTHOR (S):
                         K.; Paules, Richard S.
                         Growth Control and Cancer Group, National Institute of
CORPORATE SOURCE:
                         Environmental Health Sciences, Research Triangle Park,
                         NC, 27709, USA
                         Environmental Health Perspectives Supplements (1999),
SOURCE:
                         107(1), 5-24
                         CODEN: EHPSEO; ISSN: 1078-0475
                         National Institute of Environmental Health Sciences
PUBLISHER:
                         Journal; General Review
DOCUMENT TYPE:
                         English
LANGUAGE:
     A review with 490 refs. The ability of cells to maintain genomic
     integrity is vital for cell survival and proliferation. Lack of fidelity
     in DNA replication and maintenance can result in deleterious mutations
     leading to cell death or, in multicellular organisms, cancer. The purpose
     of this review is to discuss the known signal transduction pathways that
     regulate cell cycle progression and the mechanisms cells employ to insure
     DNA stability in the face of genotoxic stress. In particular, we focus on
     mammalian cell cycle checkpoint functions, their role in maintaining DNA
     stability during the cell cycle following exposure to genotoxic agents,
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and the gene products that act in checkpoint function signal transduction

. Aliei

1 - - 21

cascades. Key transitions in the cell cycle are regulated by the activities of various protein kinase complexes composed of cyclin and cyclin-dependent kinase (Cdk) mols. Surveillance control mechanisms that check to ensure proper completion of early events and cellular integrity before initiation of subsequent events in cell cycle progression are referred to as cell cycle checkpoints and can generate a transient delay that provides the cell more time to repair damage before progressing to the next phase of the cycle. A variety of cellular responses are elicited that function in checkpoint signaling to inhibit cyclin/Cdk activities. These responses include the p53-dependent and p53-independent induction of Cdk inhibitors and the p53-independent inhibitory phosphorylation of Cdk mols. themselves. Eliciting proper G1, S, and G2 checkpoint responses to double-strand DNA breaks requires the function of the Ataxia telangiectasia mutated gene product. Several human heritable cancer-prone syndromes known to alter DNA stability have been found to have defects in checkpoint surveillance pathways. Exposures to several common sources of genotoxic stress, including oxidative stress, ionizing radiation, UV radiation, and the genotoxic compound benzo[a]pyrene, elicit cell cycle checkpoint responses that show both similarities and differences in their mol. signaling.

CC 4-0 (Toxicology)

Section cross-reference(s): 8, 14

Robert Day Com

REFERENCE COUNT:

490 THERE ARE 490 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE

FORMAT

L214 ANSWER 12 OF 22 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2006:38566 HCAPLUS

DOCUMENT NUMBER: 144:485602

TITLE: Increased transferrin receptor expression following

11q23 deletion as a mechanism of malignant progression

in chronic lymphocytic leukemia

AUTHOR(S): Shackelford, Rodney E.; Bhalodia, Ami R.;

Cotelingam, James D.; Veillon, Diana M.;

Lowery-Nordberg, Mary

CORPORATE SOURCE: Department of Pathology, Louisiana State University at

Shreveport, LA, 711030-3932, USA

SOURCE: Medical Hypotheses (2005), Volume Date 2006, 66(3),

509-512

CODEN: MEHYDY; ISSN: 0306-9877

PUBLISHER: Elsevier Ltd.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

A review. Chronic lymphocytic leukemia (CLL) is a common adult leukemia characterized by the accumulation of mature neoplastic B-lymphocytes. Typically, CLL follows an indolent course, with most patients surviving for many years. However, 10-20% of CLL patients carry 11q23 chromosomal deletions and often exhibit a more severe disease course, with earlier onset of symptoms, shortened lymphocyte doubling time, poor response to therapy, and shortened survival. The mol. basis for 11q23 deletions resulting in a poor prognosis is currently poorly understood. The tumor suppressor gene, ataxia-telangiectasia mutated (ATM, 11q22.3-23.1), is considered a likely candidate gene whose loss could result in the poor prognosis associated with 11q23 deletion and is mutated in a significant percentage of CLL cases. Recently, recombinant ATM expression in ATM-deficient cells was found to decrease transferrin receptor (TfR) expression, suggesting that deletion of the chromosomal region carrying ATM results in increased TfR expression. TfR imports iron into cells, an event necessary for DNA synthesis and cell growth. Addnl., rapidly growing malignant cells, including lymphomas and CLL, often

express high TfR levels. Based on this, we propose that one mol. mechanism by which 11q23 deletions confer a poor prognosis in CLL is via increased TfR expression secondary to ATM loss, resulting in the increased cellular iron import, and hence increased capacity for malignant growth. Our hypothesis may also partially explain why gallium, an atomically iron-like toxic metal that binds to transferrin and the TfR is incorporated into cells and was previously demonstrated to have anti-tumor activity in patients with lymphomas refractory to other chemotherapeutic treatments.

CC 14-0 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 3

TT Proteins

RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)

(ATM (ataxia telangiectasia mutated); chromosomal

11q23 deletion may confer poor prognosis in CLL patient via increased TfR expression secondary to tumor suppressor gene ATM loss resulting in cellular iron import hence increased capacity for malignant growth)

REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS, MEDLINE, BIOSIS' - CONTINUE? (Y)/N:y

L214 ANSWER 13 OF 22 MEDLINE on STN DUPLICATE 1

ACCESSION NUMBER: 2006016513 MEDLINE DOCUMENT NUMBER: PubMed ID: 16326028

TITLE: Increased transferrin receptor expression following 11q23

deletion as a mechanism of malignant progression in chronic

lymphocytic leukemia.

AUTHOR: Shackelford Rodney E; Bhalodia Ami R; Cotelingam

James D; Veillon Diana M; Lowery-Nordberg Mary

CORPORATE SOURCE: Louisiana State University at Shreveport, Department of

Pathology, 1501 Kings Hwy, P.O. Box 33932, Shreveport, LA

711030-3932, USA.. RdnyShac@aol.com

SOURCE: Medical hypotheses, (2006) Vol. 66, No. 3, pp. 509-12.

Electronic Publication: 2005-12-02.
Journal code: 7505668. ISSN: 0306-9877.

PUB. COUNTRY: Scotland: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200606

ENTRY DATE: Entered STN: 11 Jan 2006

Last Updated on STN: 23 Jun 2006 Entered Medline: 22 Jun 2006

ABSTRACT:

Chronic lymphocytic leukemia (CLL) is a common adult leukemia characterized by the accumulation of mature neoplastic B-lymphocytes. Typically, CLL follows an indolent course, with most patients surviving for many years. However, 10-20% of CLL patients carry 11q23 chromosomal deletions and often exhibit a more severe disease course, with earlier onset of symptoms, shortened lymphocyte doubling time, poor response to therapy, and shortened survival. The molecular basis for 11q23 deletions resulting in a poor prognosis is currently poorly understood. The tumor suppressor gene, ataxia-telangiectasia mutated (ATM, 11q22.3-23.1), is considered a likely candidate gene whose loss could result in the poor prognosis associated with 11q23 deletion and is

161740

mutated in a significant percentage of CLL cases. Recently, recombinant ATM expression in ATM-deficient cells was found to decrease transferrin receptor (TfR) expression, suggesting that deletion of the chromosomal region carrying ATM results in increased TfR expression. TfR imports iron into cells, an event necessary for DNA synthesis and cell growth. Additionally, rapidly growing malignant cells, including lymphomas and CLL, often express high TfR levels. Based on this, we propose that one molecular mechanism by which 11q23 deletions confer a poor prognosis in CLL is via increased TfR expression secondary to ATM loss, resulting in the increased cellular iron import, and hence increased capacity for malignant growth. Our hypothesis may also partially explain why gallium, an atomically iron-like toxic metal that binds to transferrin and the TfR is incorporated into cells and was previously demonstrated to have anti-tumor activity in patients with lymphomas refractory to other chemotherapeutic treatments.

CONTROLLED TERM:

B-Lymphocytes: PA, pathology

Cell Cycle Proteins: GE, genetics

Chromosome Deletion

*Chromosomes, Human, Pair 11

DNA-Binding Proteins: GE, genetics

Disease Progression

*Gene Deletion

Humans

11,000 1100

*Leukemia, Lymphocytic, Chronic: GE, genetics

Lymphocytes: ME, metabolism

Prognosis

Protein-Serine-Threonine Kinases: GE, genetics

*Receptors, Transferrin: BI, biosynthesis Receptors, Transferrin: GE, genetics Recombinant Proteins: ME, metabolism Tumor Suppressor Proteins: GE, genetics

CHEMICAL NAME:

O (Cell Cycle Proteins); O (DNA-Binding Proteins); O (Receptors, Transferrin); O (Recombinant Proteins); O

(Tumor Suppressor Proteins); EC 2.7.1.37

(Protein-Serine-Threonine Kinases); EC 2.7.1.37 (

ataxia telangiectasia mutated protein)

L214 ANSWER 14 OF 22 MEDLINE ON STN ACCESSION NUMBER: 2000144083 MEDLINE

DOCUMENT NUMBER:

PubMed ID: 10677503

TITLE:

The catalytic subunit of DNA-dependent protein kinase selectively regulates p53-dependent apoptosis but not

cell-cycle arrest.

AUTHOR: Wang S; Guo M; Ouyang H; Li X; Cordon-Cardo C;

Kurimasa A; Chen D J; Fuks Z; Ling C C; Li G C

CORPORATE SOURCE: Department of Medical Physics, Memorial Sloan-Kettering

Cancer Center, 1275 York Avenue, New York, NY 10021; and Los Alamos National Laboratory, Los Alamos, NM 87545, USA.

CONTRACT NUMBER:

CA-31397 (NCI) CA-56909 (NCI) CA-78497 (NCI)

+

SOURCE:

Proceedings of the National Academy of Sciences of the United States of America, (2000 Feb 15) Vol. 97, No. 4, pp.

1584-8.

Journal code: 7505876. ISSN: 0027-8424.

PUB. COUNTRY:

United States

DOCUMENT TYPE:

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE:

English

FILE SEGMENT:

Priority Journals

ENTRY MONTH:

200003

ENTRY DATE:

Entered STN: 30 Mar 2000 Last Updated on STN: 20 Apr 2002

Entered Medline: 23 Mar 2000

ABSTRACT:

DNA damage induced by ionizing radiation (IR) activates p53, leading to the regulation of downstream pathways that control cell-cycle progression and apoptosis. However, the mechanisms for the IR-induced p53 activation and the differential activation of pathways downstream of p53 are unclear. Here we provide evidence that the catalytic subunit of DNA-dependent protein kinase (DNA-PKcs) serves as an upstream effector for p53 activation in response to IR, linking DNA damage to apoptosis. DNA-PKcs knockout (DNA-PKcs-/-) mice were exposed to whole-body IR, and the cell-cycle and apoptotic responses were examined in their thymuses. Our data show that IR induction of apoptosis and Bax expression, both mediated via p53, was significantly suppressed in the thymocytes of DNA-PKcs-/- mice. In contrast, IR-induced cell-cycle arrest and p21 expression were normal. Thus, DNA-PKcs deficiency selectively disrupts p53-dependent apoptosis but not cell-cycle arrest. We also confirmed previous findings that p21 induction was attenuated and cell-cycle arrest was defective in the thymoctyes of whole body-irradiated Atm-/- mice, but the apoptotic response was unperturbed. Taken together, our results support a model in which the upstream effectors DNA-PKcs and Atm selectively activate p53 to differentially regulate cell-cycle and apoptotic responses. Whereas Atm selects for cell-cycle arrest but not apoptosis, DNA-PKcs selects for apoptosis but not cell-cycle arrest.

CONTROLLED TERM:

Animals

*Apoptosis: GE, genetics

Apoptosis: RE, radiation effects

Ataxia Telangiectasia: GE, genetics

*Cell Cycle: GE, genetics Cell Cycle: RE, radiation effects DNA Repair: RE, radiation effects

DNA-Activated Protein Kinase

*DNA-Binding Proteins

Flow Cytometry

In Situ Nick-End Labeling

Mice

Mice, Knockout

*Protein-Serine-Threonine Kinases: GE, genetics Protein-Serine-Threonine Kinases: ME, metabolism

Proto-Oncogene Proteins: ME, metabolism

*Proto-Oncogene Proteins c-bcl-2

Research Support, U.S. Gov't, Non-P.H.S. Research Support, U.S. Gov't, P.H.S.

Thymus Gland: PA, pathology

Thymus Gland: RE, radiation effects *Tumor Suppressor Protein p53: ME, metabolism

Whole-Body Irradiation

bcl-2-Associated X Protein CHEMICAL NAME:

0 (Bax protein, mouse); 0 (DNA-Binding Proteins); 0 (Proto-Oncogene Proteins); 0 (Proto-Oncogene Proteins

c-bcl-2); 0 (Tumor Suppressor Protein p53); 0

(bcl-2-Associated X Protein); EC 2.7.1.37 (DNA-Activated Protein Kinase); EC 2.7.1.37 (Protein-Serine-Threonine

Kinases)

L214 ANSWER 15 OF 22

MEDLINE on STN MEDLINE

ACCESSION NUMBER: 1998347138

DOCUMENT NUMBER: TITLE:

PubMed ID: 9682216

A model for ATM heterozygote identification in a large

population: four founder-effect ATM mutations identify most

of Costa Rican patients with ataxia

telangiectasia.

1.00

Telatar M; Wang S; Castellvi-Bel S; Tai L Q; AUTHOR:

Sheikhavandi S; Regueiro J R; Porras O; Gatti R A

andv 1

CORPORATE SOURCE: Department of Pathology, University of California at Los

Angeles School of Medicine 90095, USA.

Molecular genetics and metabolism, (1998 May) Vol. 64, No. SOURCE:

1, pp. 36-43.

Journal code: 9805456. ISSN: 1096-7192.

PUB. COUNTRY: United States

Journal; Article; (JOURNAL ARTICLE) DOCUMENT TYPE:

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199810

ENTRY DATE: Entered STN: 20 Oct 1998

Last Updated on STN: 20 Oct 1998

Entered Medline: 6 Oct 1998

ABSTRACT:

Ataxia telangiectasia (A-T) is an autosomal recessive disorder with a broad range of clinical manifestations and a frequency of 1:40,000-100,000 live births. Epidemiological studies have suggested that A-T heterozygotes are at an elevated risk of breast cancer. ATM mutations occur worldwide over the entire ATM gene, making it difficult to identify heterozygotes in large populations. However, some founder-effect mutations are specific for certain populations. Here, we present four mutations in Costa Rican A-T patients that accounted for 86-93% of 41 patients studied in two batches. We have developed assays for rapid detection of these four mutations which can be used diagnostically. They will also enable the Costa Rican population to be used as a model for analyzing the role of ATM heterozygosity in cancer development and other disorders.

CONTROLLED TERM: Ataxia Telangiectasia: DI, diagnosis *Ataxia Telangiectasia: GE, genetics

Codon, Terminator

Costa Rica

Exons: GE, genetics *Founder Effect Genes, Recessive

*Genetic Screening: MT, methods

*Haplotypes

*Heterozygote Detection

Humans

Point Mutation Restriction Mapping Sequence Deletion

0 (Codon, Terminator) CHEMICAL NAME:

L214 ANSWER 16 OF 22 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on

STN

ACCESSION NUMBER: 2006:170137 BIOSIS DOCUMENT NUMBER: PREV200600171437

TITLE: Ataxia-telangiectasia mutated gene

product activity increases resistance to Aspergillus

fumigatus gliotoxin toxicity.

AUTHOR (S): Shackelford, R. E. [Reprint Author]; Fu, Y.;

Abdelbaqi, M.; Lowery-Nordberg, M.; Chen, A.

CORPORATE SOURCE: Louisiana State Univ, Shreveport, LA 71105 USA

SOURCE: Modern Pathology, (JAN 2006) Vol. 19, No. Suppl. 1, pp.

257A-258A.

Meeting Info.: 95th Annual Meeting of the

United-States-and-Canadian-Academy-of-Pathology. Atlanta,

GA, USA. February 11 -17, 2006. US & Canadian Acad Pathol.

3...

ISSN: 0893-3952.

Conference; (Meeting) DOCUMENT TYPE:

Conference; Abstract; (Meeting Abstract)

LANGUAGE:

English

ENTRY DATE:

Entered STN: 9 Mar 2006

Last Updated on STN: 9 Mar 2006

CONCEPT CODE:

General biology - Symposia, transactions and proceedings

00520

Genetics - Plant 03504

Biochemistry studies - General 10060 Toxicology - General and methods 22501

51522 Plant physiology - Chemical constituents

INDEX TERMS:

Major Concepts

Toxicology; Biochemistry and Molecular Biophysics

INDEX TERMS:

Chemicals & Biochemicals

ATM; gliotoxin

ORGANISM:

Classifier Fungi Imperfecti or Deuteromycetes 15500

Super Taxa

Fungi; Plantae Organism Name

Aspergillus fumigatus (species)

Taxa Notes

Fungi, Microorganisms, Nonvascular Plants, Plants

REGISTRY NUMBER:

GENE NAME:

67-99-2 (gliotoxin) Aspergillus fumigatus ATM gene [Aspergillus fumigatus

ataxia telangiectasia mutated gene] (Fungi Imperfecti or Deuteromycetes)

L214 ANSWER 17 OF 22 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on

STN

ACCESSION NUMBER:

2005:414873 BIOSIS PREV200510210084

DOCUMENT NUMBER: TITLE:

The ataxia-telangiectasia gene product

is required for genomic stability following labile ferric

iron exposure.

Shackelford, R. E. [Reprint Author]; Manuszak, R. AUTHOR (S):

P.; Wang, S.; Lowery-Norberg, M.; Chen, A.

CORPORATE SOURCE:

SOURCE:

Louisiana State Univ, Shreveport, LA 71105 USA Modern Pathology, (JAN 2005) Vol. 18, No. Suppl. 1, pp.

301A.

Meeting Info.: 94th Annual Meeting of the

United-States-and-Canadian-Academy-of-Pathology. San

Antonio, TX, USA. February 26 -March 04, 2005. US Canadian

Acad Pathol. ISSN: 0893-3952.

Conference; (Meeting) DOCUMENT TYPE:

Conference; Abstract; (Meeting Abstract)

English LANGUAGE:

Entered STN: 19 Oct 2005 ENTRY DATE:

Last Updated on STN: 19 Oct 2005

General biology - Symposia, transactions and proceedings CONCEPT CODE:

00520

Genetics - General 03502 Genetics - Human 03508

Biochemistry studies - General 10060

Cardiovascular system - Blood vessel pathology 14508

Nervous system - Pathology 20506

Immunology - Immunopathology, tissue immunology 34508 11104. 3

INDEX TERMS:

Major Concepts '

Molecular Genetics (Biochemistry and Molecular

Biophysics)

INDEX TERMS:

Diseases

ataxia-telangiectasia: vascular

disease, genetic disease, immune system disease, nervous

system disease

Ataxia Telangiectasia (MeSH)

INDEX TERMS:

Chemicals & Biochemicals

ferric iron; desferal: chelating
agent; apoferriton: chelating agent;

quercetin: chelating agent;
epigallocatechin-3 gallate:

chelating agent

INDEX TERMS:

Miscellaneous Descriptors

cell viability; genomic stability; ferric iron exposure 20074-52-6 (ferric iron)

REGISTRY NUMBER:

138-14-7 (desferal) 117-39-5 (quercetin)

989-51-5 (epigallocatechin-3

gallate)

GENE NAME:

human ATM gene gene (Hominidae)

L214 ANSWER 18 OF 22 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on

STN

ACCESSION NUMBER: DOCUMENT NUMBER:

2005:523626 BIOSIS PREV200510313589

TITLE:

A MAP4K4-TRF2 cycle amplifies apoptotic signals in mouse

myocardium.

AUTHOR (S):

Xie, Min [Reprint Author]; Wang, Sam C.; Zhang,

Dou; Prahash, Arun J.; Oh, Hidemasa; Sano, Motoaki; Wang, Xiaozhen; Pocius, Jennifer S.; Taffet, George E.; Michael, Lloyd H.; Tan, Tse-Hua; Entman, Mark L.; Schneider, Michael

D.

CORPORATE SOURCE:

Baylor Coll Med, Houston, TX 77030 USA

SOURCE:

Circulation, (OCT 26 2004) Vol. 110, No. 17, Suppl. S, pp.

1.

Meeting Info.: 77th Scientific Meeting of the

American-Heart-Association. New Orleans, LA, USA. November

07 -10, 2004. Amer Heart Assoc. CODEN: CIRCAZ. ISSN: 0009-7322.

DOCUMENT TYPE:

Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE:

English

ENTRY DATE:

Entered STN: 1 Dec 2005

Last Updated on STN: 1 Dec 2005

ABSTRACT:Background: Loss of TRF2 (Telomeric Repeat-binding Factor-2) causes apoptosis in cardiomyocytes and other cell types, but how it actuates apoptosis is unknown. Here, we studied the HPK/GCK-like Kinase (HGK, MAP4K4) and its potential relation to telomere dysfunction in cardiomyocytes. Results: (1) In mouse myocardium, each of four biological signals for cardiomyocyte apoptosis induced the kinase activity of HGK: biomechanical stress, ischemia/ reperfusion injury, and gain-of-function mutations for TNF alpha or Gq. (2) Analogous results were seen in cultured cardiomyocytes, using oxidative stress, ceramide, doxorubicin, and Gq. (3) HGK-induced apoptosis occurred via TAK1 (MAP3K7), JNK, and dissipation of mitochondrial membrane potential. (4) Interrupting normal TRF2 function in cardiomyocytes with dnTRF2 elicited phosphorylation of p53 and histone H2AX, targets for the ataxia-telangiectasia mutated (ATM) DNA damage signaling pathway. (5) Evidence placing TRF2 upstream of the apoptotic HGK-TAK1-JNK signaling module includes: dnTRF2 or knockdown of TRF2

by antisense oligos activated HGK in cardiomyocytes and/or mice; TRF2 reduced basal HGK activity, and apoptosis due to dnTRF2 was largely blocked by dnTAK1 or dnJNK1. (6) Activation of the HGK-TAK1-JNK pathway, in turn, markedly reduced TRF2 levels. (7) Conversely, dominant-negative mutations of the kinases protected TRF2 loss induced by ceramide, a potent activator of HGK. (8) To test the predicted function of TRF2 in vivo, we created alpha MHC driven gain-of-function and dominant-inhibitory (cm mutations. As anticipated, the dnTRF2 transgene caused HGK activation, myocardial apoptosis, dysfunction, and premature mortality in mice. (9) Conversely, wild-type TRF2 conferred protection against the apoptotic cardiomyopathy provoked by doxorubicin. Conclusions: Activation of the MAP4K, HGK, is a highly generalizable response to pro-apoptotic stress signals in cultured cardiomyocytes and the intact heart. Because loss of TRF2 activates the HGK pathway and activation of the HGK pathway induces TRF2 loss, our findings suggest that loss of TRF2 is reciprocally coupled to activity of the HGK death pathway, as a positive feedback loop that amplifies apoptotic signals.

CONCEPT CODE:

General biology - Symposia, transactions and proceedings

00520

Cytology - Animal 02506

Biochemistry studies - General 10060

Biochemistry studies - Proteins, peptides and amino acids

Biochemistry studies - Lipids 10066

Cardiovascular system - Physiology and biochemistry 14504

Endocrine - General 17002

Muscle - Physiology and biochemistry 17504

INDEX TERMS:

Major Concepts

Cardiovascular System (Transport and Circulation)

INDEX TERMS:

Parts, Structures, & Systems of Organisms

heart: circulatory system; cardiomyocyte: muscular

system, circulatory system; myocardium: muscular system,

circulatory system

INDEX TERMS:

Chemicals & Biochemicals

TNF-alpha [tumor necrosis factor-alpha]; p53; ceramide; doxorubicin; H2AX; telomeric repeat-binding factor-2

[TRF2]; HPK-GCK-like kinase [MAP4K4]

INDEX TERMS:

Miscellaneous Descriptors

oxidative stress; biomechanical stress; premature

mortality

ORGANISM:

Classifier

Muridae 86375

Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Organism Name mouse (common)

Taxa Notes

Animals, Chordates, Mammals, Nonhuman Vertebrates,

Nonhuman Mammals, Rodents, Vertebrates

REGISTRY NUMBER:

104404-17-3 (ceramide) 23214-92-8 (doxorubicin)

L214 ANSWER 19 OF 22 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on

STN

ACCESSION NUMBER:

2003:476393 BIOSIS

PREV200300476393

DOCUMENT NUMBER: TITLE:

Functional expression of ATM gene carried by HSV amplicon

vector in vitro and in vivo.

AUTHOR (S):

Qi, Jianguo [Reprint Author]; Manuszak, Ryan [Reprint

Author]; Shackelford, Rodney [Reprint Author];

Cheng, Dong [Reprint Author]; Smith, Michael [Reprint

```
Author]; Link, Charles J. Jr. [Reprint Author]; Wang, Suming [Reprint Author]
```

CORPORATE SOURCE:

SOURCE:

Stoddard Cancer Research Institute, Des Moines, IA, USA Proceedings of the American Association for Cancer Research

Annual Meeting, (July 2003) Vol. 44, pp. 1097. print. Meeting Info.: 94th Annual Meeting of the American

Association for Cancer Research. Washington, DC, USA. July

11-14, 2003. ISSN: 0197-016X.

DOCUMENT TYPE:

Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE:

English

, 1... 11

ENTRY DATE:

Entered STN: 15 Oct 2003

Last Updated on STN: 15 Oct 2003

CONCEPT CODE:

General biology - Symposia, transactions and proceedings

00520

Cytology - Animal 02506 Genetics - General 03502 Genetics - Animal 03506

Biochemistry studies - Proteins, peptides and amino acids

10064

Cardiovascular system - Physiology and biochemistry 14504

Cardiovascular system - Blood vessel pathology 14508 Nervous system - Physiology and biochemistry 20504 Nervous system - Pathology 20506

Nervous system - Pathology 20506 Genetics of bacteria and viruses 31500 Virology - General and methods 33502

Immunology - Immunopathology, tissue immunology 34508

INDEX TERMS:

Major Concepts

Cardiovascular System (Transport and Circulation); Molecular Genetics (Biochemistry and Molecular Biophysics); Nervous System (Neural Coordination)

INDEX TERMS:

Parts, Structures, & Systems of Organisms

caudate: nervous system; neuron: nervous system;

putamen: nervous system

INDEX TERMS:

Diseases

Ataxia-telangiectasia: genetic

disease, immune system disease, nervous system disease,

vascular disease, genetics
Ataxia Telangiectasia (MeSH)

INDEX TERMS:

Chemicals & Biochemicals

ataxia-telangiectasia cDNA [
ataxia-telangiectasia complementary

DNA]

INDEX TERMS:

Methods & Equipment

Western blot: genetic techniques, laboratory techniques; gamma-ray irradiation: clinical techniques, therapeutic and prophylactic techniques; immunohistochemistry:

immunologic techniques, laboratory techniques Miscellaneous Descriptors

INDEX TERMS:

cell survival rate; oxidative stress

ORGANISM: Classifier

Herpesviridae 03115

Super Taxa

dsDNA Viruses; Viruses; Microorganisms

Organism Name

Herpes simplex virus type-1 (common) [Human herpesvirus

1 (species)]: gene vector

Taxa Notes

Double-Stranded DNA Viruses, Microorganisms, Viruses

ORGANISM:

Classifier

Muridae 86375

Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Organism Name

rat (common): adult

Taxa Notes

Animals, Chordates, Mammals, Nonhuman Vertebrates,

Nonhuman Mammals, Rodents, Vertebrates

GENE NAME:

rat ATM gene (Muridae): expression, open reading frame

L214 ANSWER 20 OF 22 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on

STN

ACCESSION NUMBER: 2002:395524 BIOSIS DOCUMENT NUMBER: PREV200200395524

TITLE:

ATM-dependent gene expression changes after different forms

of DNA damage.

AUTHOR(S):

Heinloth, Alexandra N. [Reprint author]; Shackelford, Rodney E. [Reprint author]; Innes, Cynthia L. [Reprint author]; Amin, Rupesh P. [Reprint author]; Sieber, Stella G. [Reprint author]; Flores, Kristina G. [Reprint author]; Bennett, Lee [Reprint author]; Bushel, Pierre R. [Reprint

author]; Paules, Richard S. [Reprint author]

CORPORATE SOURCE:

National Institute of Environmental Health Sciences,

Research Triangle Park, NC, USA

SOURCE:

Proceedings of the American Association for Cancer Research

Annual Meeting, (March, 2002) Vol. 43, pp. 626. print. Meeting Info.: 93rd Annual Meeting of the American

Association for Cancer Research. San Francisco, California,

USA. April 06-10, 2002.

ISSN: 0197-016X.

DOCUMENT TYPE:

Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE:

English

ENTRY DATE:

Entered STN: 24 Jul 2002

Last Updated on STN: 29 Aug 2002

CONCEPT CODE:

General biology - Symposia, transactions and proceedings

00520

Genetics - General 03502 Genetics - Human 03508

Biochemistry studies - Nucleic acids, purines and

pyrimidines 10062

Cardiovascular system - Physiology and biochemistry 14508 Cardiovascular system - Blood vessel pathology Integumentary system - Physiology and biochemistry

Nervous system - Physiology and biochemistry 20504

Nervous system - Pathology 20506

Immunology - Immunopathology, tissue immunology

INDEX TERMS:

Major Concepts Cardiovascular System (Transport and Circulation); Integumentary System (Chemical Coordination and Homeostasis); Molecular Genetics (Biochemistry and Molecular Biophysics); Nervous System (Neural

Coordination)

Diseases

INDEX TERMS:

Parts, Structures, & Systems of Organisms

dermal diploid fibroblast: integumentary system

INDEX TERMS:

ataxia telangiectasia: genetic

disease, immune system disease, nervous system disease,

vascular disease, genetics

24.406

Ataxia Telangiectasia (MeSH)

INDEX TERMS:

Chemicals & Biochemicals

DNA; RNA; ataxia telangiectasia

mutated [ATM]: expression; cDNA [complementary DNA];
cyclin E-associated kinase; serine/threonine kinase;

t-butyl-hydroperoxide

INDEX TERMS:

Miscellaneous Descriptors

DNA damage; UV radiation [ultraviolet radiation];

oxidative stress; Meeting Abstract

REGISTRY NUMBER:

9026-43-1 (SERINE/THREONINE KINASE)

GENE NAME:

human ATM gene [human ataxia

telangiectasia mutated gene] (Hominidae):

expression

L214 ANSWER 21 OF 22 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on

STN

ACCESSION NUMBER:

1999:177572 BIOSIS PREV199900177572

DOCUMENT NUMBER: TITLE:

The ataxia telangiectasia gene product

is required for oxidative stress-induced G1 and G2

checkpoint functions in human fibroblasts.

AUTHOR(S): Shackelford, Rodney E.; Innes, Cynthia L.;

Sieber, Stella O.; Paules, Richard S.

CORPORATE SOURCE:

Natl. Inst. Environ. Health Sci., P.O. Box 12233, Research

Triangle Park, NC 27709, USA

SOURCE:

Proceedings of the American Association for Cancer Research

Annual Meeting, (March, 1999) Vol. 40, pp. 742. print. Meeting Info.: 90th Annual Meeting of the American

Association for Cancer Research. Philadelphia,

Pennsylvania, USA. April 10-14, 1999. American Association

for Cancer Research. ISSN: 0197-016X.

DOCUMENT TYPE:

Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE:

English

ENTRY DATE: Entered STN: 5 May 1999

Last Updated on STN: 5 May 1999

CONCEPT CODE:

Genetics - Human 03508

General biology - Symposia, transactions and proceedings

00520

INDEX TERMS:

Major Concepts

Genetics

INDEX TERMS:

Parts, Structures, & Systems of Organisms

fibroblasts

INDEX TERMS:

Diseases

ataxia telangiectasia: genetic

disease, immune system disease, nervous system disease,

vascular disease

Ataxia Telangiectasia (MeSH)

INDEX TERMS:

Chemicals & Biochemicals

reactive oxygen species; human ataxia

telangiectasia gene

INDEX TERMS:

Miscellaneous Descriptors

oxidative stress; G-1 checkpoint function; G-2

checkpoint function; Meeting Abstract

REGISTRY NUMBER:

7782-44-7 (OXYGEN)

L214 ANSWER 22 OF 22 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on

STN

ACCESSION NUMBER: 1997:231253 BIOSIS

DOCUMENT NUMBER:

PREV199799530456

TITLE:

Characterization of ATM expression in cell cycle

checkpoints and cellular senescence.

AUTHOR (S):

Afshari, C. A.; Innes, C. L.; Cable, P. L.; Shackelford, R.; Xu, G.; Hill, D.; Paules, R. S.

CORPORATE SOURCE:

National Inst. Environmental Health Sciences, Research

Triangle Park, NC 27709, USA

SOURCE:

Proceedings of the American Association for Cancer Research

Annual Meeting, (1997) Vol. 38, No. 0, pp. 157.

Meeting Info.: Eighty-eighth Annual Meeting of the American Association for Cancer Research. San Diego, California,

USA. April 12-16, 1997.

ISSN: 0197-016X.

DOCUMENT TYPE:

Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE:

English

ENTRY DATE:

Entered STN: 2 Jun 1997

Last Updated on STN: 9 Jul 1997

CONCEPT CODE:

Cytology - Human 02508 Genetics - Human 03508

Biochemistry studies - Proteins, peptides and amino acids

10064

Biochemistry studies - Carbohydrates 10068

Biophysics - Molecular properties and macromolecules

10506

Biophysics - Membrane phenomena 10508

Cardiovascular system - Blood vessel pathology 14508

Nervous system - Pathology 20506

Immunology - Immunopathology, tissue immunology 34508

INDEX TERMS:

Major Concepts

Biochemistry and Molecular Biophysics; Cardiovascular System (Transport and Circulation); Cell Biology; Genetics; Immune System (Chemical Coordination and Homeostasis); Membranes (Cell Biology); Nervous System

(Neural Coordination)

INDEX TERMS:

Chemicals & Biochemicals

KINASE

INDEX TERMS:

Miscellaneous Descriptors

ANTIBODIES; ATAXIA TELANGIECTASIA;

ATAXIA TELANGIECTASIA GENE;

ATAXIA TELANGIECTASIA MUTANT PROTEIN;

CARDIOVASCULAR SYSTEM; CELL BIOLOGY; CELL CYCLE CHECKPOINTS; CELLULAR SENESCENCE; CHROMOSOME

ABNORMALITIES; EXPRESSION; FIBROBLASTS; GENETIC DISEASE;

GENETICS; IMMUNE SYSTEM; IMMUNE SYSTEM DISEASE;

MUTATION; NERVOUS SYSTEM DISEASE; NUCLEAR PROTEIN; PI-3

KINASE FAMILY; VASCULAR DISEASE

REGISTRY NUMBER:

9031-44-1 (KINASE)

Напоч

=> 🗆

=> file hcaplus FILE 'HCAPLUS' ENTERED AT 15:30:14 ON 17 JUL 2006 USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT. PLEASE SEE "HELP USAGETERMS" FOR DETAILS. COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

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FILE COVERS 1907 - 17 Jul 2006 VOL 145 ISS 4 FILE LAST UPDATED: 16 Jul 2006 (20060716/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

'OBI' IS DEFAULT SEARCH FIELD FOR 'HCAPLUS' FILE

=> d que L54

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L2	1	SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
L3	1	SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
L4	1	SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
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L6	2	SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
L7	6	SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
		OR L6)
L8	1	SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
L9	1	SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
L10	1	SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT)"/CN
L11	1	SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN
L12	0	SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
L13	1	SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
		OR "DEFEROXAMINE METHANESULFONATE"/CN)
L14	0	SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
L15	1	SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
L16	1	SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
L17	1	SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
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L19	1	SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
L20	1	SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
L21	6	SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
L22	4	SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
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L47
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                   OR "DEFEROXAMINE METHANESOLFONATE / CN/

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1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN

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4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
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                        OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
                        L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
                    1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
22 SEA FILE=REGISTRY ABB=ON PLU=ON (117-39-5/BI OR 146426-40-6/B
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                          OR 480-18-2/BI OR 480-40-0/BI OR 480-41-1/BI OR 482-39-3/BI
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L55
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L56
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=> d que L71

17794

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L46	8	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	LOUIS-BAR/BI		
L47	0	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	CEREBELLO OCULOCUTANEOUS		
		TELANGIECT?/BI						
L48	0	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	CEREBELLO OCULOT?/BI -		
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L71	3	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	L69 AND L70		

=> s (L54 or L56 or L71) not L208

L215 3 (L54 OR L56 OR L71) NOT L208

=> file medline

FILE 'MEDLINE' ENTERED AT 15:30:19 ON 17 JUL 2006

FILE LAST UPDATED: 15 JUL 2006 (20060715/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 is now (26 Feb.) available. For details on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>). See also:

http://www.nlm.nih.gov/mesh/

http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html

http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html

http://www.nlm.nih.gov/pubs/techbull/nd05/nd05 2006 MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the

. . .

MeSH 2006 vocabulary.

L97

This file contains CAS Registry Numbers for easy and accurate substance identification.

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L6
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L7
                          OR L6)
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^{\text{L8}}
                       1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
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1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN
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L12
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L13
                         OR "DEFEROXAMINE METHANESULFONATE"/CN)
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L14
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1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN

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L22
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                           OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
                           L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
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24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
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3932 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA

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92986 SEA FILE=MEDLINE ABB=ON PLU=ON CHELATING AGENTS+NT/CT

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19528 SEA FILE=MEDLINE ABB=ON PLU=ON IRON CHELATING AGENTS+NT/CT

1267 SEA FILE=MEDLINE ABB=ON PLU=ON SIDEROPHORES/CT

6055 SEA FILE=MEDLINE ABB=ON PLU=ON SIDEROPHORES+NT/CT

SEL PLU=ON L31 1 - CHEM : 255 TERMS
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L85
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L90
L91
L92
L93
                                                                           255 TERMS
                           SEL PLU=ON L31 1- CHEM:
L94
                68933 SEA FILE=MEDLINE ABB=ON PLU=ON L94
L95
                       7 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND ((L88 OR L89 OR L90
L96
                          OR L91 OR L92 OR L93) OR L95)
=> d que L98
                 2457 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA/CT
L83
                3932 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA
L84
                3935 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA (2A) TELANGIECTASIA
3935 SEA FILE=MEDLINE ABB=ON PLU=ON (L83 OR L84 OR L85)
L85
L86
                37091 SEA FILE=MEDLINE ABB=ON PLU=ON CHELAT?
```

=> d que L105

```
1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
L1
              1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
L2
                                            PLU=ON FERRIOXAMINE B C?/CN
L3
              1 SEA FILE=REGISTRY ABB=ON
                                            PLU=ON FERRIOXAMINE B H?/CN
              1 SEA FILE=REGISTRY ABB=ON
L4
                                                    FERRIOXAMINE B M?/CN
L5
              1 SEA FILE=REGISTRY ABB=ON
                                            PLU=ON
                                                    FERRIOXAMINE B P?/CN
              2 SEA FILE=REGISTRY ABB=ON
                                            PLU=ON
L6
                                                    (L1 OR L2 OR L3 OR L4 OR L5
              6 SEA FILE=REGISTRY ABB=ON
                                            PLU=ON
L7
                OR L6)
              1 SEA FILE=REGISTRY ABB=ON
                                            PLU=ON CP 94/CN
L8
              1 SEA FILE=REGISTRY ABB=ON
                                            PLU=ON
                                                    EDTA/CN
L9
                                           PLU=ON
                                                    "EDTA (CHELATING AGENT)"/CN
              1 SEA FILE=REGISTRY ABB=ON
L10
                                           PLU=ON
                                                    DEFEROXAMINE B MESYLATE/CN
              1 SEA FILE=REGISTRY ABB=ON
L11
              O SEA FILE=REGISTRY ABB=ON PLU=ON
                                                    L11 AND L7
L12
             1 SEA FILE=REGISTRY ABB=ON PLU=ON
                                                    ("DEFEROXAMINE MESYLATE"/CN
L13
                OR "DEFEROXAMINE METHANESULFONATE"/CN)
              O SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
L14
             1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
L15
             1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
L16
             1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
L17
             1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
L18
             1 SEA FILE=REGISTRY ABB=ON PLU=ON
                                                    DTPA/CN
L19
             1 SEA FILE=REGISTRY ABB=ON PLU=ON
                                                    PENICILLAMINE/CN
L20
              6 SEA FILE=REGISTRY ABB=ON PLU=ON
                                                    BATHOCUP?/CN
L21
              4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
L22
                C?/CN
             23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L23
                OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
                L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
              1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
L29
             24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
L31
          2457 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA/CT
3932 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA
3935 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA (2A) TELANGIECTASIA
L83
L84
L85
          3935 SEA FILE=MEDLINE ABB=ON PLU=ON (L83 OR L84 OR L85)
13204 SEA FILE=MEDLINE ABB=ON PLU=ON CHELATING AGENTS/CT
L86
L88
          92986 SEA FILE=MEDLINE ABB=ON PLU=ON CHELATING AGENTS+NT/CT
L89
          3231 SEA FILE=MEDLINE ABB=ON PLU=ON IRON CHELATING AGENTS/CT
L90
          19528 SEA FILE=MEDLINE ABB=ON PLU=ON IRON CHELATING AGENTS+NT/CT
L91
          1267 SEA FILE=MEDLINE ABB=ON PLU=ON SIDEROPHORES/CT 6055 SEA FILE=MEDLINE ABB=ON PLU=ON SIDEROPHORES+NT/CT
L92
L93
                SEL PLU=ON L31 1- CHEM: 255 TERMS
L94
          68933 SEA FILE=MEDLINE ABB=ON PLU=ON L94
L95
              7 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND ((L88 OR L89 OR L90
                 OR L91 OR L92 OR L93) OR L95)
          61053 SEA FILE=MEDLINE ABB=ON PLU=ON ANTIOXID?
L100
          18939 SEA FILE=MEDLINE ABB=ON PLU=ON FLAV!NOID?/BI
L101
          32882 SEA FILE=MEDLINE ABB=ON PLU=ON FLAVONOIDS+NT/CT
L102
                QUE ABB=ON PLU=ON TRANSITION ELEMENTS+NT/CT
L103
              3 SEA FILE=MEDLINE ABB=ON PLU=ON L96 AND ((L100 OR L101 OR
L105
                L102 OR L103))
```

=> d que L108

L1 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN

```
1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
L2
                  1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
L3
                  1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
L4
                  1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
L5
                  2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
L6
                  6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L7
                     OR L6)
                  1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
L8
                  1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
L9
                  1 SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT)"/CN
L10
                  1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN
L11
                  O SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
L12
                  1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
L13
                     OR "DEFEROXAMINE METHANESULFONATE"/CN)
                  O SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
L14
                  1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
L15
               1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
L16
L17
L18
L19
L20
L21
                 4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
L22
                     C?/CN
                 23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L23
                     OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
                     L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
                  1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
L29
                 24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
L31
                 22 SEA FILE=REGISTRY ABB=ON PLU=ON (117-39-5/BI OR 146426-40-6/B
L37
                     I OR 153-18-4/BI OR 17912-87-7/BI OR 446-72-0/BI OR 480-16-0/BI
                       OR 480-18-2/BI OR 480-40-0/BI OR 480-41-1/BI OR 482-39-3/BI
                     OR 490-46-0/BI OR 491-70-3/BI OR 491-80-5/BI OR 50-78-2/BI OR
                     520-26-3/BI OR 520-27-4/BI OR 520-33-2/BI OR 520-36-5/BI OR
                     522-12-3/BI OR 525-82-6/BI OR 577-85-5/BI OR 989-51-5/BI)
                  1 SEA FILE=REGISTRY ABB=ON PLU=ON 50-78-2
L38
                 21 SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38
            21 SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38

2457 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA/CT

3932 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA

3935 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA (2A) TELANGIECTASIA

3935 SEA FILE=MEDLINE ABB=ON PLU=ON (L83 OR L84 OR L85)

13204 SEA FILE=MEDLINE ABB=ON PLU=ON CHELATING AGENTS/CT

92986 SEA FILE=MEDLINE ABB=ON PLU=ON CHELATING AGENTS+NT/CT

3231 SEA FILE=MEDLINE ABB=ON PLU=ON IRON CHELATING AGENTS+NT/CT

19528 SEA FILE=MEDLINE ABB=ON PLU=ON IRON CHELATING AGENTS+NT/CT

1267 SEA FILE=MEDLINE ABB=ON PLU=ON SIDEROPHORES/CT

6055 SEA FILE=MEDLINE ABB=ON PLU=ON SIDEROPHORES/CT

SEL PLU=ON L31 1 - CHEM : 255 TERMS
L39
L83
L84
L85
L86
L88
L89
L90
L91
L92
L93
L94
                     SEL PLU=ON L31 1- CHEM:
                                                               255 TERMS
             68933 SEA FILE=MEDLINE ABB=ON PLU=ON L94
L95
                   7 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND ((L88 OR L89 OR L90
L96
                     OR L91 OR L92 OR L93) OR L95)
                     SEL PLU=ON L39 1- CHEM:
                                                                344 TERMS
L106
             19788 SEA FILE=MEDLINE ABB=ON PLU=ON L106
L107
                  2 SEA FILE=MEDLINE ABB=ON PLU=ON L107 AND L96
L108
```

=> d que L119

2457 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA/CT 3932 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA L83 L84

```
3935 SEA FILE-MEDLINE ABB-ON PLU-ON ATAXIA (2A) TELANGIECTASIA
L85
          3935 SEA FILE=MEDLINE ABB=ON PLU=ON (L83 OR L84 OR L85)
L86
               QUE ABB=ON PLU=ON FERRIOXAMIN? OR DEFEROXAMIN? OR DESF
L109
               ERROXAMIN? OR DEFERRIOXAMIN?
               QUE ABB=ON PLU=ON EDETIC ACID/CT
L110
               QUE ABB=ON PLU=ON CP94
L111
               QUE ABB=ON PLU=ON HYDROXAMIC ACIDS/CT
L112
               QUE ABB=ON PLU=ON APOFERRITIN/CT
L113
               QUE ABB=ON PLU=ON CDTA
L114
               QUE ABB=ON PLU=ON DTPA OR PENTATIC ACID
L115
               QUE ABB=ON PLU=ON PENICILLAMINE
L116
               QUE ABB=ON PLU=ON
L117
                                   BATHOCUPROINE
               QUE ABB=ON PLU=ON BATHOCUPROIN
L118
             6 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND (L109 OR L110 OR L111
L119
               OR L112 OR L113 OR L114 OR L115 OR L116 OR L117 OR L118)
```

=> s (L96 or L98 or L105 or L108 or L119) not L209

L216 7 (L96 OR L98 OR L105 OR L108 OR L119) NOT L209

=> file embase

. 1742

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FILE COVERS 1974 TO 17 Jul 2006 (20060717/ED)

EMBASE has been reloaded. Enter HELP RLOAD for details.

 ${\tt EMBASE}$ is now updated daily. SDI frequency remains weekly (default) and biweekly.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> d que L139

L1	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	FERRIOXAMINE/CN
L2	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	FERRIOXAMINE B/CN
L3	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	FERRIOXAMINE B C?/CN
L4	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	FERRIOXAMINE B H?/CN
L5	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	FERRIOXAMINE B M?/CN
L6	2	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	FERRIOXAMINE B P?/CN
L7	6	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	(L1 OR L2 OR L3 OR L4 OR L5
		OR I	L6)			
L8	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	CP 94/CN
L9	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	EDTA/CN
L10	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	"EDTA (CHELATING AGENT)"/CN
L11	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	DEFEROXAMINE B MESYLATE/CN
L12	0	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	L11 AND L7
L13	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	("DEFEROXAMINE MESYLATE"/CN
		OR '	DEFEROXAMINE N	METHANES	JLFONATE	"/CN)
L14	0	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	L13 AND L7
L15	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	DESFERAL/CN
L16	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	DESFERAL M?/CN
L17	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	APOFERRITIN?/CN
L18	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	CDTA/CN
L19	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	DTPA/CN
L20	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	PENICILLAMINE/CN

11/06/2006

```
6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
L21
                    4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
L22
                        C?/CN
                   23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L23
                        OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
                        L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
                     1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
L29
                   24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
L31
                2332 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA+UF/CT
L128
                3044 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA
L129
                  62 SEA FILE=EMBASE ABB=ON PLU=ON LOUIS BAR
L130
                     2 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTATICA
L131
                     O SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLOOCULOCU
L132
                        TANEA
                     O SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLO
L133
                        OCULOCUTANEA
                3053 SEA FILE=EMBASE ABB=ON PLU=ON (L128 OR L129 OR L130 OR L131
L134
                        OR L132 OR L133)
               98621 SEA FILE=EMBASE ABB=ON PLU=ON CHELATING AGENT+NT/CT
L136
                        SEL PLU=ON L31 1- CHEM: 255 TERMS
L137
               61283 SEA FILE=EMBASE ABB=ON PLU=ON L137
L138
                   14 SEA FILE=EMBASE ABB=ON PLU=ON L134 AND (L136 OR L138)
L139
=> d que L142
                     1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
L1
                     1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
L2
                    1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L3
L4
L5
L6
L7
            OK L6)

1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT)"/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN

0 SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7

1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN

OR "DEFEROXAMINE METHANESIII.FONATE"/CN)
L8
L9
L10
L11
L12
L13
                      OR "DEFEROXAMINE METHANESULFONATE"/CN)
            OR "DEFEROXAMINE METHANESULFONATE"/CN)

0 SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7

1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN

6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN

4 SEA FILE=PEGISTRY ABB=ON DIJI-ON DIFTUVIENCETDIAMINE
L14
L15
L16
L17
L18
L19
L20
L21
                    4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
L22
                        C?/CN
                    23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L23
                        OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
                        L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
                  1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
L29
L31
                2332 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA+UF/CT
3044 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA
62 SEA FILE=EMBASE ABB=ON PLU=ON LOUIS BAR
L128
L129
L130
                     2 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTATICA
L131
```

```
PLU=ON
                                               TELANGIECTASIA CEREBELLOOCULOCU
L132
             O SEA FILE=EMBASE ABB=ON
               TANEA
                                               TELANGIECTASIA CEREBELLO
             O SEA FILE=EMBASE ABB=ON
                                       PLU=ON
L133
               OCULOCUTANEA
          3053 SEA FILE=EMBASE ABB=ON
                                       PLU=ON (L128 OR L129 OR L130 OR L131
L134
               OR L132 OR L133)
L136
         98621 SEA FILE=EMBASE ABB=ON
                                       PLU=ON CHELATING AGENT+NT/CT
                                              255 TERMS
L137
               SEL PLU=ON L31 1- CHEM :
L138
         61283 SEA FILE=EMBASE ABB=ON
                                       PLU=ON L137
            14 SEA FILE=EMBASE ABB=ON
                                       PLU=ON
                                               L134 AND (L136 OR L138)
L139
                                       PLU=ON
         .25033 SEA FILE=EMBASE ABB=ON
                                               FLAVONOID+NT/CT
L140
         35447 SEA FILE=EMBASE ABB=ON
                                       PLU=ON ANTIOXIDANT+NT/CT
L141
             4 SEA FILE=EMBASE ABB=ON PLU=ON L139 AND (L140 OR L141)
L142
=> d que L145
```

T 3	-	OF THE DEGLOSEY ADD ON DILL ON DEDDLOVANTNE/ON
L1		SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
L2		,
L3		SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
L4		SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
L5		SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
L6		SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
L7	6	SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
		OR L6)
L8		SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
L9		SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
L10		SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT)"/CN
L11		SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN
L12		SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
L13	1	SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
		OR "DEFEROXAMINE METHANESULFONATE"/CN)
L14	0	SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
L15	1	SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
L16	1	SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
L17	1	SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
L18	1	SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
L19	1	SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
L20	1	SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
L21	6	SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
L22	4	SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
		C?/CN
L23	23	SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
		OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
		L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
L29	1	SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
L31	24	SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
L37	22	SEA FILE=REGISTRY ABB=ON PLU=ON (117-39-5/BI OR 146426-40-6/B
		I OR 153-18-4/BI OR 17912-87-7/BI OR 446-72-0/BI OR 480-16-0/BI
		OR 480-18-2/BI OR 480-40-0/BI OR 480-41-1/BI OR 482-39-3/BI
		OR 490-46-0/BI OR 491-70-3/BI OR 491-80-5/BI OR 50-78-2/BI OR
		520-26-3/BI OR 520-27-4/BI OR 520-33-2/BI OR 520-36-5/BI OR
		522-12-3/BI OR 525-82-6/BI OR 577-85-5/BI OR 989-51-5/BI)
L38	1	SEA FILE=REGISTRY ABB=ON PLU=ON 50-78-2
L39		SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38
L128		SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA+UF/CT
L129		SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA
L130		SEA FILE=EMBASE ABB=ON PLU=ON LOUIS BAR
L131		SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTATICA
L132		SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLOOCULOCU
1176	U	TIBE-ENDAGE ADD-ON FEG-ON TELIANGTECTASIA CEREBELLOOCOLOCO

```
TANEA
             O SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLO
L133
               OCULOCUTANEA
          3053 SEA FILE=EMBASE ABB=ON PLU=ON (L128 OR L129 OR L130 OR L131
L134
               OR L132 OR L133)
         98621 SEA FILE=EMBASE ABB=ON PLU=ON CHELATING AGENT+NT/CT
L136
               SEL PLU=ON L31 1- CHEM :
                                           255 TERMS
L137
         61283 SEA FILE=EMBASE ABB=ON PLU=ON L137
L138
            14 SEA FILE=EMBASE ABB=ON PLU=ON L134 AND (L136 OR L138)
L139
               SEL PLU=ON L39 1- CHEM: 344 TERMS
L143
         24101 SEA FILE=EMBASE ABB=ON PLU=ON L143
L144
             2 SEA FILE=EMBASE ABB=ON PLU=ON L139 AND L144
L145
=> d que L150
          2332 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA+UF/CT
L128
          3044 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA
L129
            62 SEA FILE=EMBASE ABB=ON PLU=ON LOUIS BAR
L130
             2 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTATICA
L131
             O SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLOOCULOCU
L132
               TANEA
             O SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLO
L133
               OCULOCUTANEA
          3053 SEA FILE=EMBASE ABB=ON PLU=ON (L128 OR L129 OR L130 OR L131
L134
               OR L132 OR L133)
         31221 SEA FILE=EMBASE ABB=ON PLU=ON CHELAT?
L149
             4 SEA FILE=EMBASE ABB=ON PLU=ON L149 AND L134
L150
```

=> s (L139 or L142 or l145 or L150) not L210

L217 10 (L139 OR L142 OR L145 OR L150) NOT L210

=> file biosis

FILE 'BIOSIS' ENTERED AT 15:30:29 ON 17 JUL 2006 Copyright (c) 2006 The Thomson Corporation

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RECORDS LAST ADDED: 12 July 2006 (20060712/ED)

=> d que L168

L1	1 SEA FILE=REGISTRY ABB=ON	PLU=ON FERRIOXAMINE/CN
L2	1 SEA FILE=REGISTRY ABB=ON	PLU=ON FERRIOXAMINE B/CN
L3	1 SEA FILE=REGISTRY ABB=ON	PLU=ON FERRIOXAMINE B C?/CN
L4	1 SEA FILE=REGISTRY ABB=ON	PLU=ON FERRIOXAMINE B H?/CN
L5	1 SEA FILE=REGISTRY ABB=ON	PLU=ON FERRIOXAMINE B M?/CN
L6	2 SEA FILE=REGISTRY ABB=ON	PLU=ON FERRIOXAMINE B P?/CN
L7	6 SEA FILE=REGISTRY ABB=ON	PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
	OR L6)	
L8	1 SEA FILE=REGISTRY ABB=ON	PLU=ON CP 94/CN
L9	1 SEA FILE=REGISTRY ABB=ON	PLU=ON EDTA/CN
L10	1 SEA FILE=REGISTRY ABB=ON	PLU=ON "EDTA (CHELATING AGENT)"/CN
L11	1 SEA FILE=REGISTRY ABB=ON	PLU=ON DEFEROXAMINE B MESYLATE/CN

an

```
O SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
L12
             1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
L13
               OR "DEFEROXAMINE METHANESULFONATE"/CN)
             O SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
L14
             1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
L15
             1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
L16
            1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
L17
            1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
L18
            1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
L19
            1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
L20
             6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
L21
             4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
L22
               C?/CN
            23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L23
               OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
               L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
             1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
L29
            24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
L31
          3180 SEA FILE=BIOSIS ABB=ON PLU=ON ATAXIA TELANGIECTASIA
L155
         38028 SEA FILE=BIOSIS ABB=ON PLU=ON CHELAT?
L157
L158
               SEL PLU=ON L31 1- CHEM :
                                             255 TERMS
         59051 SEA FILE=BIOSIS ABB=ON PLU=ON L158
L159
          3182 SEA FILE=BIOSIS ABB=ON PLU=ON ATAXIA TELANGIECTASIA?
L160
          3396 SEA FILE=BIOSIS ABB=ON PLU=ON' SIDEROPHOR?
L163
            42 SEA FILE=BIOSIS ABB=ON PLU=ON SIDEROCHROM?
L164
            72 SEA FILE=BIOSIS ABB=ON PLU=ON LOUIS BAR
L165
          3223 SEA FILE=BIOSIS ABB=ON PLU=ON L155 OR L160 OR L165
L166
               QUE ABB=ON PLU=ON L157 OR (L163 OR L164) OR L159
L167
             7 SEA FILE=BIOSIS ABB=ON PLU=ON L166 AND L167
L168
```

=> d que L172

```
1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
L1
                1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L2
L3
L4
L5
L6
L7
                   OR L6)
                1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
L8
                1 SEA FILE=REGISTRY ABB=ON PLU=ON
                                                            EDTA/CN
Ŀ9
                                                            "EDTA (CHELATING AGENT)"/CN
                1 SEA FILE=REGISTRY ABB=ON
                                                   PLU=ON
L10
                1 SEA FILE=REGISTRY ABB=ON
                                                   PLU=ON
                                                            DEFEROXAMINE B MESYLATE/CN
L11
L12
                O SEA FILE=REGISTRY ABB=ON
                                                   PLU=ON
                                                            L11 AND L7
                                                            ("DEFEROXAMINE MESYLATE"/CN
L13
                1 SEA FILE=REGISTRY ABB=ON
                                                  PLU=ON
                   OR "DEFEROXAMINE METHANESULFONATE"/CN)
L14
                O SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
                1 SEA FILE=REGISTRY ABB=ON PLU=ON
                                                            DESFERAL/CN
L15
                1 SEA FILE=REGISTRY ABB=ON
                                                  PLU=ON
                                                            DESFERAL M?/CN
L16
L17
                1 SEA FILE=REGISTRY ABB=ON
                                                  PLU=ON
                                                            APOFERRITIN?/CN
                1 SEA FILE=REGISTRY ABB=ON
                                                  PLU=ON
                                                            CDTA/CN
L18
                1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
L19
                1 SEA FILE=REGISTRY ABB=ON
                                                  PLU=ON
                                                            PENICILLAMINE/CN
L20
                6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
L21
                4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
L22
                   C?/CN
L23
               23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
                   OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
```

```
L15 OR I:16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
                         1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
L29
                        24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
L31
                        22 SEA FILE=REGISTRY ABB=ON PLU=ON (117-39-5/BI OR 146426-40-6/B
L37
                              I OR 153-18-4/BI OR 17912-87-7/BI OR 446-72-0/BI OR 480-16-0/BI
                               OR 480-18-2/BI OR 480-40-0/BI OR 480-41-1/BI OR 482-39-3/BI
                              OR 490-46-0/BI OR 491-70-3/BI OR 491-80-5/BI OR 50-78-2/BI OR
                              520-26-3/BI OR 520-27-4/BI OR 520-33-2/BI OR 520-36-5/BI OR
                              522-12-3/BI OR 525-82-6/BI OR 577-85-5/BI OR 989-51-5/BI)
                          1 SEA FILE=REGISTRY ABB=ON PLU=ON 50-78-2
L38
                        21 SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38
L39
                    3180 SEA FILE=BIOSIS ABB=ON PLU=ON ATAXIA TELANGIECTASIA
L155
                  38028 SEA FILE=BIOSIS ABB=ON PLU=ON CHELAT?
L157
                              SEL PLU=ON L31 1- CHEM:
                                                                                        255 TERMS
L158
                  59051 SEA FILE=BIOSIS ABB=ON PLU=ON L158
L159
                    3182 SEA FILE=BIOSIS ABB=ON PLU=ON ATAXIA TELANGIECTASIA?
L160
                    3396 SEA FILE=BIOSIS ABB=ON PLU=ON SIDEROPHOR?
L163
                       42 SEA FILE=BIOSIS ABB=ON PLU=ON SIDEROCHROM?
L164
                       72 SEA FILE=BIOSIS ABB=ON PLU=ON LOUIS BAR
L165
                    3223 SEA FILE=BIOSIS ABB=ON PLU=ON L155 OR L160 OR L165
L166
                              OUE ABB=ON PLU=ON L157 OR (L163 OR L164) OR L159
L167
                          7 SEA FILE-BIOSIS ABB-ON PLU-ON L166 AND L167
L168
L170
                             SEL PLU=ON L39 1- CHEM: 344 TERMS
                  31206 SEA FILE=BIOSIS ABB=ON PLU=ON L170
L171
                          3 SEA FILE=BIOSIS ABB=ON PLU=ON L171 AND L168
L172
=> d que L173
                          1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
L1
                          1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
L2
                          1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
L3
                        1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C:/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H:/CN
1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M:/CN
2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P:/CN
6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L4
L5
L6
L7
                              OR L6)
                          1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
L8
                        1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
L9
                    1 SEA FILE-REGISTRY ABB-ON PLU-ON "EDTA (CHELATING AGENT)"/CN
1 SEA FILE-REGISTRY ABB-ON PLU-ON DEFEROXAMINE B MESYLATE/CN
0 SEA FILE-REGISTRY ABB-ON PLU-ON L11 AND L7
L10
L11
L12
                        1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
L13
                            OR "DEFEROXAMINE METHANESULFONATE"/CN)
                       0 SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
L14
                       U SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7

1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN

1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN

CEA FILE=REGISTRY ABB=ON PLU=ON DESIGNATION CONTACTOR 
L15
L16
L17
L18
L19
L20
                         6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
L21
                         4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
L22
                              C?/CN
                        23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L23
                              OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
                              L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
                          1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
L29
                        24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
L31
                    3180 SEA FILE=BIOSIS ABB=ON PLU=ON ATAXIA TELANGIECTASIA
L155
```

Tarry 10/ [1

```
38028 SEA FILE=BIOSIS ABB=ON PLU=ON CHELAT?
L157
               SEL PLU=ON L31 1- CHEM:
                                             255 TERMS
L158
L159
         59051 SEA FILE=BIOSIS ABB=ON PLU=ON L158
          3182 SEA FILE=BIOSIS ABB=ON
                                      PLU=ON
                                              ATAXIA TELANGIECTASIA?
L160
          4049 SEA FILE=BIOSIS ABB=ON PLU=ON
                                              TRANSITION METAL?
L161
           160 SEA FILE=BIOSIS ABB=ON
                                      PLU=ON
                                              TRANSITION ELEM?
L162
          3396 SEA FILE=BIOSIS ABB=ON
                                      PLU=ON
                                              SIDEROPHOR?
L163
L164
            42 SEA FILE=BIOSIS ABB=ON
                                      PLU=ON
                                              SIDEROCHROM?
                                      PLU=ON
L165
            72 SEA FILE=BIOSIS ABB=ON
                                              LOUIS BAR
L166
          3223 SEA FILE=BIOSIS ABB=ON
                                      PLU=ON L155 OR L160 OR L165
               QUE ABB=ON PLU=ON L157 OR (L163 OR L164) OR L159
L167
L168
             7 SEA FILE=BIOSIS ABB=ON PLU=ON L166 AND L167
             O SEA FILE=BIOSIS ABB=ON PLU=ON L168 AND (L161 OR L162)
L173
```

=> s (L168 or L172 or L173) not L211

L218 3 (L168 OR L172 OR L173) NOT L211

=> file uspatfull

FILE 'USPATFULL' ENTERED AT 15:30:33 ON 17 JUL 2006 CA INDEXING COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

FILE COVERS 1971 TO PATENT PUBLICATION DATE: 13 Jul 2006 (20060713/PD) FILE LAST UPDATED: 13 Jul 2006 (20060713/ED) HIGHEST GRANTED PATENT NUMBER: US7076805 HIGHEST APPLICATION PUBLICATION NUMBER: US2006156447 CA INDEXING IS CURRENT THROUGH 11 Jul 2006 (20060711/UPCA) ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 13 Jul 2006 (20060713/PD) REVISED CLASS FIELDS (/NCL) LAST RELOADED: Feb 2006 USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Feb 2006

=> d que L185

L1	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	FERRIOXAMINE/CN
L2	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	FERRIOXAMINE B/CN
L3	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	FERRIOXAMINE B C?/CN
L4	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	FERRIOXAMINE B H?/CN
L5	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	FERRIOXAMINE B M?/CN
L6	2	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	FERRIOXAMINE B P?/CN
L7	6	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	(L1 OR L2 OR L3 OR L4 OR L5
		OR I	L6)			
L8	1	SÉA	FILE=REGISTRY	ABB=ON	PLU=ON	CP 94/CN
L9	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	EDTA/CN
L10	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	"EDTA (CHELATING AGENT)"/CN
L11	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	DEFEROXAMINE B MESYLATE/CN
L12	0	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	L11 AND L7
L13	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	("DEFEROXAMINE MESYLATE"/CN
		OR	DEFEROXAMINE N	METHANESU	JLFONATE	"/CN)
L14	0	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	L13 AND L7
L15	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	DESFERAL/CN
L16	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	DESFERAL M?/CN
L17	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	APOFERRITIN?/CN
L18	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	CDTA/CN
L19	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	DTPA/CN
L20	1	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	PENICILLAMINE/CN
L21	6	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	BATHOCUP?/CN
L22	4	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	DIETHYLENETRIAMINE PENTAACETI

C?/CN

```
23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L23
               OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
               L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
             1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
L29
            24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
L31
          5870 SEA FILE=USPATFULL ABB=ON PLU=ON L31
L183
          2355 SEA FILE-USPATFULL ABB-ON PLU-ON ATAXIA TELANGIECTASIA
L184
             6 SEA FILE-USPATFULL ABB-ON PLU-ON L183 AND L184
L185
=> d que L187
             1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
L1
             1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
L2
             1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
L3
             1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
L4
             1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
L5
             2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
L6
             6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L7
               OR L6)
             1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
L8
             1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
L9
             1 SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT)"/CN
L10
             1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN
L11
             O SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
L12
             1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
L13
               OR "DEFEROXAMINE METHANESULFONATE"/CN)
             O SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
L14
            1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
L15
            1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
L16
            1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
L17
            1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
L18
            1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
L19
            1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
L20
             6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
L21
             4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
L22
               C?/CN
            23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
L23
               OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
               L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
             1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
L29
            24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
L31
            22 SEA FILE=REGISTRY ABB=ON PLU=ON (117-39-5/BI OR 146426-40-6/B
L37
               I OR 153-18-4/BI OR 17912-87-7/BI OR 446-72-0/BI OR 480-16-0/BI
                OR 480-18-2/BI OR 480-40-0/BI OR 480-41-1/BI OR 482-39-3/BI
               OR 490-46-0/BI OR 491-70-3/BI OR 491-80-5/BI OR 50-78-2/BI OR
               520-26-3/BI OR 520-27-4/BI OR 520-33-2/BI OR 520-36-5/BI OR
               522-12-3/BI OR 525-82-6/BI OR 577-85-5/BI OR 989-51-5/BI)
             1 SEA FILE=REGISTRY ABB=ON PLU=ON 50-78-2
L38
            21 SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38
L39
           5870 SEA FILE=USPATFULL ABB=ON PLU=ON L31
L183
          2355 SEA FILE-USPATFULL ABB-ON PLU-ON ATAXIA TELANGIECTASIA
L184
             6 SEA FILE=USPATFULL ABB=ON PLU=ON L183 AND L184
L185
          1795 SEA FILE=USPATFULL ABB=ON PLU=ON L39
L186
             1 SEA FILE-USPATFULL ABB-ON PLU-ON L185 AND L186
L187
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^{=&}gt; s (L185 or L187) not 1212

4.301

L219

5 (L185 OR L187) NOT L212

=> file wpix

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http://www.stn-international.de/stndatabases/details/dwpi_r.html <<<
'BIX' IS DEFAULT SEARCH FIELD FOR 'WPIX' FILE</pre>

=> d que L197

L188 247 SEA FILE=WPIX ABB=ON PLU=ON ATAXIA TELANGIECTASIA/BIX
L195 5533 SEA FILE=WPIX ABB=ON PLU=ON (RAAMBT/DCN OR RAGNQ8/DCN OR
RA0DFA/DCN OR RA0EMC/DCN OR RA0JBK/DCN OR RA00TF/DCN OR
RA0055/DCN OR RA021P/DCN OR RA0529/DCN OR RA1HHQ/DCN OR
RA1XA5/DCN OR RA37W9/DCN OR R00064/DCN OR R00195/DCN OR
R00268/DCN OR R00971/DCN OR R01179/DCN OR R01318/DCN OR
R01319/DCN OR R03811/DCN OR R03812/DCN OR R03949/DCN OR
R04870/DCN OR R06069/DCN OR R06174/DCN OR R06413/DCN OR
R06747/DCN OR R07001/DCN OR R07027/DCN OR R08105/DCN OR
R08504/DCN OR R09163/DCN OR R09222/DCN OR R09884/DCN OR
R11605/DCN OR R19085/DCN OR R19452/DCN OR R20811/DCN OR
R22037/DCN)
L197 2 SEA FILE=WPIX ABB=ON PLU=ON L188 AND L195

=> s L197 not L213

L220 2 L197 NOT L213

=> => dup rem L215 L216 L217 L218 L219 L220

FILE 'HCAPLUS' ENTERED AT 15:31:21 ON 17 JUL 2006

USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.

PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

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FILE 'MEDLINE' ENTERED AT 15:31:21 ON 17 JUL 2006

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FILE 'BIOSIS' ENTERED AT 15:31:21 ON 17 JUL 2006

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FILE 'WPIX' ENTERED AT 15:31:21 ON 17 JUL 2006 COPYRIGHT (C) 2006 THE THOMSON CORPORATION

PROCESSING COMPLETED FOR L215 PROCESSING COMPLETED FOR L216 PROCESSING COMPLETED FOR L217 PROCESSING COMPLETED FOR L218 PROCESSING COMPLETED FOR L219 PROCESSING COMPLETED FOR L220

24 DUP REM L215 L216 L217 L218 L219 L220 (6 DUPLICATES REMOVED)

ANSWERS '1-3' FROM FILE HCAPLUS ANSWERS '4-9' FROM FILE MEDLINE ANSWERS '10-16' FROM FILE EMBASE ANSWER '17' FROM FILE BIOSIS ANSWERS '18-22' FROM FILE USPATFULL

ANSWERS '23-24' FROM FILE WPIX

=> d ibib abs hitind hitstr L221 1-3; d iall L221 4-17; d ibib abs kwic hitstr L221 18-22; d iall ind L221 23-24

L221 ANSWER 1 OF 24 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2

ACCESSION NUMBER:

2005:1214439 HCAPLUS

DOCUMENT NUMBER:

144:18854

TITLE:

Tachpyridine, a metal chelator, induces G2 cell-cycle

arrest, activates checkpoint kinases, and sensitizes

cells to ionizing radiation

AUTHOR (S):

Turner, JoLyn; Koumenis, Constantinos; Kute, Timothy E.; Planalp, Roy P.; Brechbiel, Martin W.; Beardsley, Dillon; Cody, Brooke; Brown, Kevin D.; Torti, Frank

M.; Torti, Suzy V.

CORPORATE SOURCE:

Department of Biochemistry, Wake Forest University

Health Sciences, Durham, NH, USA Blood (2005), 106(9), 3191-3199

SOURCE: PUBLISHER:

CODEN: BLOOAW; ISSN: 0006-4971 American Society of Hematology

DOCUMENT TYPE:

Journal English

LANGUAGE: Iron is critical for cell growth and proliferation. Iron chelators are being explored for a number of clin. applications, including the treatment of neurodegenerative disorders, heart disease, and cancer. To uncover mechanisms of action of tachpyridine, a chelator currently undergoing preclin. evaluation as an anticancer agent, cell-cycle anal. was performed. Tachpyridine arrested cells at G2, a radiosensitive phase of the cell cycle, and enhanced the sensitivity of cancer cells but not nontransformed cells to ionizing radiation. G2 arrest was p53 independent and was accompanied by activation of the checkpoint kinases CHK1 and CHK2. G2 arrest was blocked by UCN-01, a CHK1 inhibitor, but proceeded in CHK2 knock-out cells, indicating a critical role for CHK1 in G2 arrest. Tachpyridine-induced cell-cycle arrest was abrogated in cells treated with caffeine, an inhibitor of the ataxia-telangiectasia mutated/ataxia-telangiectasia-mutated and Rad3-related (ATM/ATR) kinases. Further, G2 arrest proceeded in ATM-deficient cells but was blocked in ATR-deficient cells, implicating ATR as the proximal kinase in tachpyridine-mediated G2 arrest. Collectively, our results suggest that iron chelators may function as antitumor and radioenhancing agents and uncover a previously unexplored activity of iron chelators in

11/06/2006

```
activation of ATR and checkpoint kinases.
```

CC 8-9 (Radiation Biochemistry)

IT Proteins

RL: BSU (Biological study, unclassified); BIOL (Biological study)

(ATM (ataxia telangiectasia mutated); metal

chelator tachpyridine as antitumor radiosensitizer and effect on cell cycle and checkpoint kinases)

IT Antitumor agents

Chelating agents

Human

Radiosensitizers, biological

Radiotherapy

(metal chelator tachpyridine as antitumor radiosensitizer and effect on cell cycle and checkpoint kinases)

REFERENCE COUNT:

83 THERE ARE 83 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L221 ANSWER 2 OF 24 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER:

2004:266901 HCAPLUS

DOCUMENT NUMBER:

140:302341

TITLE:

Protein complexes of the tumor necrosis factor- α signalling pathway for diagnosis, therapy and drug

screening

INVENTOR(S):

Bouwmeester, Tewis; Huhse, Bettina; Bauch, Angela;

Ruffner, Heinz; Bauer, Andreas; Kruse, Ulrich;

Kuester, Bernhard; Superti-Furga, Guilio

PATENT ASSIGNEE(S):

Cellzome Ag, Germany Eur. Pat. Appl., 549 pp.

SOURCE:

CODEN: EPXXDW

DOCUMENT TYPE:

Patent

LANGUAGE:

English

FAMILY ACC. NUM. COUNT:

PATENT INFORMATION:

PA'	TENT	NO.			KIN	D	DATE			APPL	ICAT	ION 1	NO.		D	ATE	
EP	1403	282			A1	-	2004	0331		 EP 2	002-	2180	 9		20	00209	926
	R:	AT,	BE,	CH,	DE,	DK,	ES,	FR,	GB,	GR,	IT,	LI,	LU,	NL,	SE,	MC,	PT,
		ΙE,	SI,	LT,	LV,	FI,	RO,	MK,	CY,	AL,	TR,	ВG,	CZ,	EE,	SK		
WO	2004	0357	83		A2		2004	0429	,	WO 2	003-	EP50	655		20	00309	924
WO	2004	0357	83		C2	,	2004	0930									
WO	2004	0357	83		A 3		2004	1111									
	W:	ΑE,	AG,	AL,	AM,	ΑT,	AU,	ΑZ,	BA,	BB,	BG,	BR,	BY,	ΒZ,	CA,	CH,	CN,
		CO,	CR,	CU,	CZ,	DE,	DK,	DM,	DZ,	EC,	EE,	EG,	ES,	FI,	GB,	GD,	GE,
		GH,	GM,	HR,	ΗU,	ID,	IL,	IN,	IS,	JP,	KE,	KG,	ΚP,	KR,	KZ,	LC,	LK,
		LR,	LS,	LT,	LU,	LV,	MA,	MD,	MG,	MK,	MN,	MW,	MX,	MZ,	NI,	NO,	NZ,
		OM,	PG,	PH,	PL,	PT,	RO,	RU,	SC,	SD,	SE,	SG,	SK,	SL,	SY,	ТJ,	TM,
		TN,	TR,	TT,	TZ,	UA,	ŪĠ,	US,	UZ,	VC,	VN,	YU,	ZA,	ZM,	zw		
	RW:	GH,	GM,	KE,	LS,	MW,	ΜZ,	SD,	SL,	SZ,	TZ,	ŪG,	ZM,	ZW,	AM,	ΑZ,	BY,
		KG,	ΚZ,	MD,	RU,	ΤJ,	TM,	AT,	BE,	ВG,	CH,	CY,	CZ,	DΕ,	DK,	EE,	ES,
		FΙ,	FR,	GB,	ĢR,	HU,	ΙE,	IT,	LU,	MC,	NL,	PT,	RO,	SE,	SI,	SK,	TR,
		BF,	ВJ,	CF,	CG,	CI,	CM,	GA,	GN,	GQ,	GW,	ML,	MR,	ΝE,	SN,	TD,	TG
AU	2003	2982	61		A 1		2004	0504		AU 2	003-	2982	61		2	0030	924
PRIORIT	Y APP	LN.	INFO	.:						EP 2	002-	2180	9	7	A 2	0020	926
										EP 2	003-	1002	74	1	A 2	0030	210
										WO 2	003-	EP50	655	1	W 2	0030	924
						_					-						

AB The present invention relates to protein complexes of the Tumor necrosis factor- α -signaling pathway, component proteins of said complexes, fragments and derivs. of the component proteins and antibodies specific to the complexes. The present invention also relates to methods for use of

the complexes of the TNF- α -signaling pathway and their interaction in screening, diagnosis and therapy as well to methods of preparing the complexes. Pharmaceutical compns. comprising the protein complexes and antibodies specific to the complexes are especially useful for diagnosis and treatment of inflammation, infection, neurodegenerative disease and cancer.

IC ICM C07K014-705

ICS C07K014-715; C07K016-28; C07K017-00; A61K038-17; G01N033-50

CC 15-5 (Immunochemistry)

Section cross-reference(s): 1, 3, 9, 63

IT Proteins

RL: ARU (Analytical role, unclassified); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)

(ataxia-telangiectasia group D-associated; protein

complexes of the tumor necrosis factor- α signalling pathway and antibodies for drug screening and for diagnosis and therapy)

IT 60-00-4, EDTA, biological studies 75-12-7, Formamide, biological studies 1185-53-1, Tris hydrochloride 9003-39-8, PVP 9042-14-2, Dextran sulfate

RL: ARU (Analytical role, unclassified); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study)

(protein complexes of the tumor necrosis factor- α signalling pathway and antibodies for drug screening and for diagnosis and therapy of inflammation, infection, neurodegenerative disease and cancer)

IT 60-00-4, EDTA, biological studies

RL: ARU (Analytical role, unclassified); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study) (protein complexes of the tumor necrosis factor-α signalling

pathway and antibodies for drug screening and for diagnosis and therapy of inflammation, infection, neurodegenerative disease and cancer)

RN 60-00-4 HCAPLUS

CN Glycine, N,N'-1,2-ethanediylbis[N-(carboxymethyl)- (9CI) (CA INDEX NAME)

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L221 ANSWER 3 OF 24 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2002:397930 HCAPLUS

DOCUMENT NUMBER: 136:374807

TITLE: Cosmetic or pharmaceutical composition based on lipoic

acid and pyruvic acid

INVENTOR(S): Gianfranco de Paoli, Ambrosi PATENT ASSIGNEE(S): General Topics S.R.L., Italy

SOURCE: Ital., 20 pp. CODEN: ITXXBY

DOCUMENT TYPE: Patent LANGUAGE: Italian

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
IT 1299623	В1	20000324	IT 1998-BS10	19980223

PRIORITY APPLN. INFO.:

IT 1998-BS10 199802

AB The invention concerns a composition for cosmetic or pharmaceutical use which contains as active ingredients at least lipoic acid (both reduced form and dehydrolipoic acid) and pyruvic acid, their salts, esters, and amides and stereoisomers. Each may be present in amts. from 0.0001 to 90% weight/weight

IC ICM A61K

IT

CC 63-6 (Pharmaceuticals)

Section cross-reference(s): 62

IT Nervous system, disease

(ataxia telangiectasia; cosmetic or pharmaceutical composition based on lipoic acid and pyruvic acid)

50-21-5, 2-Hydroxypropanoic acid, biological studies 50-81-7, Ascorbic acid, biological studies 52-90-4, Cysteine, biological studies 56-40-6, Glycine, biological studies 56-41-7, Alanine, biological 56-45-1, Serine, biological studies 56-84-8, Aspartic acid, 56-85-9, Glutamine, biological studies biological studies Glutamic acid, biological studies · 56-87-1, Lysine, biological studies 57-10-3, Palmitic acid, biological studies 57-11-4, Octadecanoic acid, biological studies 58-85-5, Biotin 59-30-3, Folic acid, biological 59-43-8, Thiamine, biological studies 59-67-6, Niacin, studies biological studies 60-00-4, Ethylenediaminetetraacetic acid, biological studies 60-18-4, Tyrosine, biological studies Linoleic acid, biological studies 61-90-5, Leucine, biological studies 63-68-3, Methionine, biological studies 63-91-2, Phenylalanine, 66-72-8, Pyridoxal biological studies 65-23-6, Pyridoxine 68-26-8, 69-72-7, 2-Hydroxybenzoic acid, biological studies Retinol Asparagine, biological studies 71-00-1, Histidine, biological studies 72-19-5, Threonine, biological 72-18-4, Valine, biological studies 73-22-3, Tryptophan, biological studies 73-32-5, Isoleucine, 74-79-3, Arginine, biological studies 79-14-1, biological studies Hydroxyethanoic acid, biological studies 79-83-4, Pantothenic acid 83-88-5, Riboflavin, biological studies 87-69-4, 81-13-0, Panthenol 2,3-Dihydroxybutanedioic acid, biological studies 98-92-0, Nicotinamide 107-35-7, Taurine 112-80-1, Oleic acid, biological studies 105-45-3 114-07-8, Erythromycin 116-31-4, Retinaldehyde 123-31-9, Hydroquinone, biological studies 127-17-3, Pyruvic acid, biological studies 143-07-7, Lauric acid, 127-17-3D, Pyruvic acid, derivs. 141-97-9 biological studies 147-85-3, Proline, biological studies 150-13-0 302-79-4, Retinoic acid 373-49-9, Palmitoleic 153-18-4, Rutin 443-48-1, Metronidazol 463-40-1, Linolenic acid 464-92-6, acid 473-81-4, 2,3-Dihydroxypropanoic acid 506-32-1, Asiatic acid Arachidonic acid 526-95-4, Gluconic acid 541-50-4, biological studies 544-63-8, Myristic acid, biological studies 544-64-9, Myristoleic acid 557-59-5, Lignoceric acid 600-15-7, 2-Hydroxybutanoic acid 600-22-6 617-35-6 693-72-1, Vaccenic acid 1200-22-2, Lipoic acid 1200-22-21 693-72-1, Vaccenic acid 1200-22-2D, Lipoic acid, derivs. 3380-34-5, Triclosan 3416-24-8, Glucosamine 7235-40-7, β Carotene 6556-12-3, Glucuronic acid 7512-17-6, Acetylglucosamine 9004-61-9, Hyaluronic acid 10118-90-8, Minocycline 16830-15-2, Asiaticoside 18323-44-9, Clindamycin 18449-41-7, 29204-02-2, Gadoleic acid 34540-22-2, Madecassoside Madecassic acid 38882-78-9 55306-03-1, Sericic acid 55306-04-2, Sericoside RL: COS (Cosmetic use); PEP (Physical, engineering or chemical process); PYP (Physical process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)

(cosmetic or pharmaceutical composition based on lipoic acid and pyruvic acid)

IT 60-00-4, Ethylenediaminetetraacetic acid, biological studies 153-18-4, Rutin

RL: COS (Cosmetic use); PEP (Physical, engineering or chemical process); PYP (Physical process); THU (Therapeutic use); BIOL (Biological study);

PROC (Process); USES (Uses)

(cosmetic or pharmaceutical composition based on lipoic acid and pyruvic acid)

60-00-4 HCAPLUS RN

Glycine, N, N'-1, 2-ethanediylbis[N-(carboxymethyl)- (9CI) (CA INDEX NAME) CN

153-18-4 HCAPLUS RN

4H-1-Benzopyran-4-one, $3-[[6-O-(6-deoxy-\alpha-L-mannopyranosyl)-\beta-D-$ CN (CA glucopyranosyl]oxy]-2-(3,4-dihydroxyphenyl)-5,7-dihydroxy- (9CI) INDEX NAME)

Absolute stereochemistry. Rotation (+).

L221 ANSWER 4 OF 24

MEDLINE on STN

DUPLICATE 1

ACCESSION NUMBER:

2006242510

IN-PROCESS

AUTHOR:

PubMed ID: 16651613

DOCUMENT NUMBER:

SUMOylation attenuates sensitivity toward hypoxia- or TITLE:

desferroxamine-induced injury by modulating

adaptive responses in salivary epithelial cells.

Nguyen Ha-Van; Chen Jo-Lin; Zhong Jenny; Kim Kwang-Jin; Crandall Edward D; Borok Zea; Chen Yuan; Ann David K

Department of Molecular Pharmacology and Toxicology, CORPORATE SOURCE:

University of Southern California, Los Angeles 90033-1049,

USA.

CONTRACT NUMBER:

CA-94595 (NCI) HL-38578 (NHLBI) HL-38621 (NHLBI) HL-38658 (NHLBI) HL-62569 (NHLBI) HL-64365 (NHLBI) R01-DE-10742 (NIDCR) R01-DE-14183 (NIDCR)

SOURCE:

The American journal of pathology, (2006 May) Vol. 168, No.

Hand- 10/5

5, pp. 1452-63.

Journal code: 0370502. ISSN: 0002-9440.

PUB. COUNTRY:

United States

. 1. 1 3 .

DOCUMENT TYPE:

Journal; Article; (JOURNAL ARTICLE)

English LANGUAGE:

FILE SEGMENT:

NONMEDLINE; IN-PROCESS; NONINDEXED; Abridged Index Medicus

Journals; Priority Journals

ENTRY DATE:

Entered STN: 3 May 2006 Last Updated on STN: 23 May 2006

ABSTRACT:

Hypoxic stress activates various signal transduction pathways including posttranslational modification with the ubiquitin-like SUMO protein (SUMOylation). However, the molecular mechanisms by which SUMOylation regulates hypoxic responses remain unclear. Here, we investigated the ability of rat salivary Pa-4 epithelial cells to resist cell injury elicited by 1% O(2) - or hypoxia-mimetic desferroxamine (DFO) - stimulated SUMOylation processes. By using Pa-4 cells stably transduced with lenti-SUMO-1 and a cell-permeant peptide harboring SUMO-binding motif to interfere with SUMO-dependent protein-protein interactions, we demonstrate that SUMOylation augments cell survival against DFO treatment. This appeared to be partly mediated through attenuation of Protein Kinase C (PKC)-delta activation and caspase-3 cleavage, hallmarks of pro-apoptotic signaling. Intriguingly, DFO-induced phosphorylation of DNA damage marker ataxia-***telangiectasia*** -mutated protein S1981 preceded activation of PKCdelta and caspase-3. Constitutive SUMOylation facilitated 1% O(2) - or DFO-induced nuclear factor kappaB transactivation, possibly via activation of genotoxic signaling cascade. In addition, we observed transient preservation of transepithelial electrical resistance during the early stage of hypoxia (1% O(2)) as well as enhanced transepithelial electrical resistance recovery after prolonged hypoxia in SUMO-1-expressing cell monolayers. In conclusion, our results unveil a previously unrecognized mechanism by which SUMOylation and activation of ataxia-telangiectasia-mutated protein, PKCdelta, caspase-3, and nuclear factor kappaB signaling pathways modulate

salivary adaptive responses to stress in cells exposed to either 1% O(2) or DFO.

MEDLINE on STN L221 ANSWER 5 OF 24

97433222 MEDLINE

DOCUMENT NUMBER:

ACCESSION NUMBER:

PubMed ID: 9288891

Use of a postlabelling assay to examine the removal of TITLE:

radiation-induced DNA lesions by purified enzymes and human

DUPLICATE 3

cell extracts.

Weinfeld M; Lee J; Ruiqi G; Karimi-Busheri F; Chen D; **AUTHOR:**

Allalunis-Turner J

Department of Oncology, University of Alberta, Cross Cancer CORPORATE SOURCE:

Institute, Edmonton, Canada.. mweinfel@gpu.srv.ualberta.ca

Mutation research, (1997 Aug 1) Vol. 378, No. 1-2, pp. SOURCE:

127-37.

Journal code: 0400763. ISSN: 0027-5107.

PUB. COUNTRY:

Netherlands

Journal; Article; (JOURNAL ARTICLE) DOCUMENT TYPE:

English

FILE SEGMENT:

LANGUAGE:

Priority Journals

ENTRY MONTH:

199709

ENTRY DATE:

Entered STN: 8 Oct 1997

Last Updated on STN: 8 Oct 1997 Entered Medline: 25 Sep 1997

ABSTRACT:

We have used a 32P-postlabelling assay to examine the activity of purified Esherichia coli endonuclease IV, human apurinic/apyrimidinic endonuclease I and human cell-free extracts towards irradiated DNA. The assay can detect thymine glycols, 3'-phosphoglycolate groups and at least one other major lesion that has yet to be fully characterized. It was observed that endonuclease IV removed the phosphoglycolates and the uncharacterized lesion(s) suggesting that the latter are abasic sites with modified deoxyribose residues. The purified human enzyme acted only on the phosphoglycolate residues. Cell-free extract, prepared from A549 lung carcinoma cells by sonication or treatment with toluene, efficiently removed the phosphoglycolate and unknown lesions, but was less reactive towards thymine glycols. The extract was completely inactivated by heating at 60 degrees C for 10 min. Removal of the unknown product and phosphoglycolate did not require magnesium, but 1 mM EDTA did inhibit release of the latter. The cell-free extract exhibited substantially more activity towards native than heat-denatured DNA. A comparison of extracts prepared from 4 cell lines displaying a range of radiosensitivities, including ataxia telangiectasia cell line, showed that all

contained similar levels of repair activity towards the detectable lesions.

CONTROLLED TERM:

Cell Extracts Cell Survival

*DNA: ME, metabolism

DNA: RE, radiation effects

*DNA Damage

*DNA Repair

DNA, Single-Stranded: ME, metabolism DNA-(Apurinic or Apyrimidinic Site) Lyase

*Deoxyribonuclease I: ME, metabolism Deoxyribonuclease IV (Phage T4-Induced)

Electrophoresis, Polyacrylamide Gel Escherichia coli: EN, enzymology

*Escherichia coli Proteins

Gamma Rays: AE, adverse effects Glycolates: ME, metabolism

Humans

*Lyases: ME, metabolism Magnesium: PD, pharmacology Nucleic Acid Denaturation

Phosphorus Radioisotopes: ME, metabolism

Research Support, Non-U.S. Gov't Thymine: AA, analogs & derivatives

Thymine: ME, metabolism Tumor Cells, Cultured

CAS REGISTRY NO.:

13147-57-4 (phosphoglycolate); 2943-56-8 (thymine glycol); 65-71-4 (Thymine); 7439-95-4 (Magnesium); 9007-49-2 (DNA)

CHEMICAL NAME:

O (Cell Extracts); O (DNA, Single-Stranded); O (Escherichia coli Proteins); 0 (Glycolates); 0 (Phosphorus Radioisotopes); EC 3.1.21.1 (Deoxyribonuclease I); EC

3.1.21.2 (Deoxyribonuclease IV (Phage T4-Induced)); EC 3.1.21.2 (endonuclease IV, E coli); EC 4. (Lyases); EC 4.2.99.18 (DNA-(Apurinic or Apyrimidinic Site) Lyase)

L221 ANSWER 6 OF 24

MEDLINE on STN 2004083906 MEDLINE PubMed ID: 14716295

ACCESSION NUMBER: DOCUMENT NUMBER:

The EBNA-3 gene family proteins disrupt the G2/M

checkpoint.

AUTHOR: Krauer Kenia G; Burgess Andrew; Buck Marion; Flanagan

James; Sculley Tom B; Gabrielli Brian

CORPORATE SOURCE: Queensland Institute of Medical Research and Joint Oncology

Program, University of Queensland, Brisbane, Australia...

keniaK@gimr.edu.au

SOURCE: Oncogene, (2004 Feb 19) Vol. 23, No. 7, pp. 1342-53.

Journal code: 8711562. ISSN: 0950-9232.

PUB. COUNTRY: England: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200403

ENTRY DATE: Entered STN: 20 Feb 2004

Last Updated on STN: 11 Mar 2004 Entered Medline: 10 Mar 2004

ABSTRACT:

The Epstein-Barr nuclear antigens (EBNA), EBNA-3, -4 and -6, have previously been shown to act as transcriptional regulators, however, this study identifies another function for these proteins, disruption of the G2/M checkpoint. Lymphoblastoid cell lines (LCLs) treated with a G2/M initiating drug azelaic bishydroxamine (ABHA) did not show a G2/M checkpoint response, but rather they display an increase in cell death, a characteristic of sensitivity to the cytotoxic effects of the drug. Cell cycle analysis demonstrated that the individual expression of EBNA-3, -4 or -6 are capable of disrupting the G2/M checkpoint response induced by ABHA resulting in increased toxicity, whereas EBNA-2, and -5 were not. EBNA-3 gene family protein expression also disrupted the G2/M checkpoint initiated in response to the genotoxin etoposide and the S phase inhibitor hydroxyurea. The G2 arrest in response to these drugs were sensitive to caffeine, suggesting that ATM/ATR signalling in these checkpoint responses may be blocked by the EBNA-3 family proteins. The function of EBNA-3, -4 and -6 proteins appears to be more complex than anticipated and these data suggest a role for these proteins in disrupting the host cell cycle machinery.

CONTROLLED TERM:

*Cell Cycle Proteins

DNA Damage: PH, physiology

DNA-Binding Proteins

Epstein-Barr Virus Nuclear Antigens: IM, immunology *Epstein-Barr Virus Nuclear Antigens: ME, metabolism

G2 Phase: DE, drug effects . *G2 Phase: PH, physiology

Histone Deacetylases: AI, antagonists & inhibitors

Humans

Hydroxamic Acids: PD, pharmacology

Mitosis: DE, drug effects *Mitosis: PH, physiology

Precipitin Tests

Protein-Serine-Threonine Kinases: IM, immunology Protein-Serine-Threonine Kinases: ME, metabolism

Research Support, Non-U.S. Gov't Signal Transduction: PH, physiology

Tumor Suppressor Proteins

CAS REGISTRY NO.: CHEMICAL NAME:

18992-11-5 (azelaic bishydroxamic acid)

0 (Cell Cycle Proteins); 0 (DNA-Binding Proteins); 0
(Epstein-Barr Virus Nuclear Antigens); 0 (Hydroxamic Acids); 0 (Tumor Suppressor Proteins); EC 2.7.1.- (ATR protein, human); EC 2.7.1.37 (Protein-Serine-Threonine

Kinases); EC 2.7.1.37 (ataxia

telangiectasia mutated protein); EC 2.7.1.37

(checkpoint kinase 2); EC 3.5.1.- (Histone Deacetylases)

L221 ANSWER 7 OF 24

MEDLINE on STN

ACCESSION NUMBER:

2003256335 MEDLINE

DOCUMENT NUMBER:

PubMed ID: 12782595 Histone deacetylase inhibitors activate p21(WAF1)

TITLE: Histone

expression via ATM.

AUTHOR:

Ju Rong; Muller Mark T

CORPORATE SOURCE:

Department of Molecular Genetics, The Ohio State

University, Columbus, Ohio 43210, USA.

SOURCE:

Cancer research, (2003 Jun 1) Vol. 63, No. 11, pp. 2891-7.

Journal code: 2984705R. ISSN: 0008-5472.

PUB. COUNTRY:

United States

DOCUMENT TYPE:

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE:

English

FILE SEGMENT:

Priority Journals

ENTRY MONTH:

200307

ENTRY DATE:

Entered STN: 4 Jun 2003

Last Updated on STN: 1 Aug 2003 Entered Medline: 31 Jul 2003

ABSTRACT:

Histone deacetylase (HDAC) inhibitors are known to induce expression of genes such as p21(WAF1), thereby, leading to cell cycle arrest. In this work, we show that p21(WAF1) induction by HDAC inhibitors (depsipeptide and trichostatin A) is defective in Ataxia telangiectasia (AT) cells but normal in matched wild-type (WT) cells (human diploid fibroblasts). To verify the role of ATM in this effect, we show that ectopic expression of the WT ATM gene in an AT cell line fully restores p21(WAF1) induction by the HDAC inhibitors. Furthermore, because caffeine and wortmannin attenuate p21(WAF1) induction in WT cells, it is probable that the phosphatidylinositol 3'-kinase activity is essential for this process. Besides the p21(WAF1) promoter, activation of topoisomerase IIIalpha and SV40 promoters by the HDAC inhibitors are also decreased in the AT cell lines relative to WT cells; thus, these findings pertain to other promoters. Finally, despite the obvious induction deficiency of gene expression, the overall levels of H3 and H4 histone acetylation appear to be the same between AT and normal cells in response to Taken together, the data indicate that ATM is HDAC inhibitor treatments. involved in histone acetylation-mediated gene regulation. 1-Phosphatidylinositol 3-Kinase: ME, metabolism CONTROLLED TERM:

Ataxia Telangiectasia: PA, pathology

Cell Cycle Proteins

Cyclin-Dependent Kinase Inhibitor p21

*Cyclins: BI, biosynthesis Cyclins: GE, genetics DNA-Binding Proteins

*Depsipeptides

Acetylation

Enzyme Inhibitors: PD, pharmacology

Gene Expression Regulation: PH, physiology

*Histone Deacetylases: AI, antagonists & inhibitors

Histones: GE, genetics Histones: ME, metabolism

Humans

Hydroxamic Acids: PD, pharmacology

Peptides, Cyclic: PD, pharmacology

Phosphorylation

Promoter Regions (Genetics): DE, drug effects
Protein-Serine-Threonine Kinases: BI, biosynthesis
Protein-Serine-Threonine Kinases: GE, genetics
*Protein-Serine-Threonine Kinases: PH, physiology

Transfection

Tumor Suppressor Proteins

CAS REGISTRY NO.: CHEMICAL NAME:

128517-07-7 (FR 901228); 58880-19-6 (trichostatin A) 0 (CDKN1A protein, human); 0 (Cell Cycle Proteins); 0 (Cyclin-Dependent Kinase Inhibitor p21); 0 (Cyclins); 0 (DNA-Binding Proteins); 0 (Depsipeptides); 0 (Enzyme Inhibitors); 0 (Histones); 0 (Hydroxamic Acids); 0 (Peptides, Cyclic); 0 (Tumor Suppressor Proteins); EC 2.7.1.137 (1-Phosphatidylinositol 3-Kinase); EC 2.7.1.37

(Protein-Serine-Threonine Kinases); EC 2.7.1.37 (

ataxia telangiectasia mutated protein);

~ . .

EC 3.5.1. - (Histone Deacetylases)

L221 ANSWER 8 OF 24 ACCESSION NUMBER: DOCUMENT NUMBER:

MEDLINE on STN 97459668 MEDLINE PubMed ID: 9315628

TITLE:

Regulation of p53 by metal ions and by antioxidants

: dithiocarbamate down-regulates p53 DNA-binding activity

by increasing the intracellular level of copper.

AUTHOR:

Verhaegh G W; Richard M J; Hainaut P

CORPORATE SOURCE:

Unit of Mechanisms of Carcinogenesis, International Agency

for Research on Cancer, Lyon, France.

SOURCE:

Molecular and cellular biology, (1997 Oct) Vol. 17, No. 10,

pp. 5699-706.

Journal code: 8109087. ISSN: 0270-7306.

PUB. COUNTRY: United States

DOCUMENT TYPE:

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT:

Priority Journals

ENTRY MONTH:

199710

ENTRY DATE:

Entered STN: 5 Nov 1997

Last Updated on STN: 17 Dec 2002 Entered Medline: 23 Oct 1997

ABSTRACT:

Mutations in the p53 tumor suppressor gene frequently fall within the specific DNA-binding domain and prevent the molecule from transactivating normal targets. DNA-binding activity is regulated in vitro by metal ions and by redox conditions, but whether these factors also regulate p53 in vivo is unclear. To address this question, we have analyzed the effect of pyrrolidine dithiocarbamate (PDTC) on p53 DNA-binding activity in cell lines expressing wild-type p53. PDTC is commonly regarded as an antioxidant, but it can also bind and transport external copper ions into cells and thus exert either pro- or antioxidant effects in different situations. We report that PDTC, but not N-acetyl-L-cysteine, down-regulated the specific DNA-binding activity of p53. Loss of DNA binding correlated with disruption of the immunologically "wild-type" p53 conformation. Using different ***chelators*** to interfere with copper transport by PDTC, we found that ***bathocuproinedisulfonic*** acid (BCS), a non-cell-permeable chelator of Cul+, prevented both copper import and p53 down-regulation. In contrast, 1,10-orthophenanthroline, a cell-permeable of Cu2+, promoted the redox activity of copper and ***chelator*** up-regulated p53 DNA-binding activity through a DNA damage-dependent pathway. We have previously reported that p53 protein binds copper in vitro in the form of Cul+ (P. Hainaut, N. Rolley, M. Davies, and J. Milner, Oncogene 10:27-32, 1995). The data reported here indicate that intracellular levels and redox activity of copper are critical for p53 protein conformation and DNA-binding activity and suggest that copper ions may participate in the physiological control of p53 function.

CONTROLLED TERM: Acetylcysteine: PD, pharmacology

*Antioxidants: PD, pharmacology

Cell Cycle

Cell Cycle Proteins

Cell Line

```
*Chelating Agents: PD, pharmacology
                      *Copper: ME, metabolism
                     Cyclin-Dependent Kinase Inhibitor p21
                     Cyclins: BI, biosynthesis
                     DNA: ME, metabolism
                     DNA Damage
                     DNA-Binding Proteins
                     Humans
                     Hydrogen Peroxide: PD, pharmacology
                     Intercalating Agents: PD, pharmacology
                     Ion Transport: DE, drug effects
                     Lipid Peroxidation
                     Oxidation-Reduction
                     Oxidative Stress
                     Phenanthrolines: PD, pharmacology
                     Protein Binding: DE, drug effects
                     Protein Conformation: DE, drug effects
                    *Protein-Serine-Threonine Kinases
                     Proteins: PH, physiology
                     Pyrrolidines: PK, pharmacokinetics
                     Research Support, Non-U.S. Gov't
                     Thiocarbamates: PK, pharmacokinetics
                    *Thiocarbamates: PD, pharmacology
                     Tumor Cells, Cultured
                     Tumor Suppressor Protein p53: BI, biosynthesis
                     Tumor Suppressor Protein p53: CH, chemistry
                     Tumor Suppressor Protein p53: DE, drug effects
                    *Tumor Suppressor Protein p53: ME, metabolism
                     Tumor Suppressor Proteins
                    14708-99-7 (ferroin); 25769-03-3 (pyrrolidine
CAS REGISTRY NO .:
                    dithiocarbamic acid); 616-91-1 (Acetylcysteine);
                    73348-75-1 (bathocuproine sulfonate); 7440-50-8
                    (Copper); 7722-84-1 (Hydrogen Peroxide); 9007-49-2 (DNA)
                    0 (Antioxidants); 0 (CDKN1A protein, human); 0
CHEMICAL NAME:
                    (Cell Cycle Proteins); 0 (Chelating Agents); 0
                    (Cyclin-Dependent Kinase Inhibitor p21); 0 (Cyclins); 0
                    (DNA-Binding Proteins); 0 (Intercalating Agents); 0
                    (Phenanthrolines); 0 (Proteins); 0 (Pyrrolidines); 0
                    (Thiocarbamates); 0 (Tumor Suppressor Protein p53); 0
                    (Tumor Suppressor Proteins); EC 2.7.1.37
                    (Protein-Serine-Threonine Kinases); EC 2.7.1.37 (
                    ataxia telangiectasia mutated protein)
L221 ANSWER 9 OF 24
                        MEDLINE on STN
                                 MEDLINE
ACCESSION NUMBER:
                    84002886
                    PubMed ID: 6616960
DOCUMENT NUMBER:
                    Abnormalities of lymphocyte locomotion in immunodeficiency
TITLE:
                    Van Epps D E; El-Naggar A; Ochs H D
AUTHOR:
CONTRACT NUMBER:
                    AI-07073 (NIAID)
                    CA 20819 (NCI)
                    RR37 (NCRR)
                    Clinical and experimental immunology, (1983 Sep) Vol. 53,
SOURCE:
                    No. 3, pp. 678-88.
                    Journal code: 0057202. ISSN: 0009-9104.
                    ENGLAND: United Kingdom
PUB. COUNTRY:
                    Journal; Article; (JOURNAL ARTICLE)
DOCUMENT TYPE:
                    English
LANGUAGE:
```

まんじょ/2003

Handy 10/617943 11/05/2006

FILE SEGMENT:

Priority Journals

ENTRY MONTH:

198311

ENTRY DATE:

Entered STN: 19 Mar 1990

Last Updated on STN: 3 Feb 1997 Entered Medline: 23 Nov 1983

ABSTRACT:

Lymphocyte and neutrophil locomotion were studied in 23 patients with well defined, primary immunodeficiencies. These included eight patients with common variable immune deficiency, three patients with X-linked agammaglobulinaemia, two patients with the Wiskott-Aldrich syndrome, three patients with ***ataxia*** telangiectasia, three patients with immunodeficiency and normal serum immunoglobulin concentrations, one patient with immune deficiency and hyper-IgM syndrome, two patients with Job syndrome and one patient with a granulocyte adherence defect. Random and stimulated lymphocyte and neutrophil migration were evaluated. C5a and casein were used to stimulate lymphocyte migration and C5a and formyl-methionyl-leucyl-phenylalanine (f-MLP) were used to stimulate neutrophil migration. Significantly depressed lymphocyte migration in response to casein and C5a was observed in patients with common variable immune deficiency, patients with immune deficiency and normal immunoglobulin concentration, and patients with Job syndrome. No consistent defect in lymphocyte locomotion was observed in the other patients studied. Neutrophil migration in response to C5a and f-MLP was depressed in Job syndrome, the patient with a granulocyte adherence defect, one of the six patients with common variable immune deficiency and none of the remaining patients. No significant correlation of skin test reactivity and lymphocyte migration was noted, but a correlation between the degree of lymphocyte proliferation in response to phytohaemagglutinin and lymphocyte migration in response to casein was observed. The results presented indicate that aberrations in lymphocyte migration occur in several types of immunodeficiency diseases and that defects in lymphocyte and neutrophil migration can occur simultaneously or totally independent of each other.

CONTROLLED TERM:

Check Tags: Female; Male

Adolescent Adult

Aged

Caseins

Chemotactic Factors

*Chemotaxis, Leukocyte

Child

Child, Preschool

Comparative Study

Complement C5

Complement C5a

Humans

Hypersensitivity, Delayed

*Immunologic Deficiency Syndromes: IM, immunology

Job's Syndrome: IM, immunology

Lymphocyte Activation

Lymphocytes: IM, immunology

Middle Aged

Mitosis

N-Formylmethionine Leucyl-Phenylalanine

Neutrophils: IM, immunology

Phytohemagglutinins: PD, pharmacology Receptors, Antigen, B-Cell: AN, analysis

Research Support, Non-U.S. Gov't

Research Support, U.S. Gov't, P.H.S.

59880-97-6 (N-Formylmethionine Leucyl-Phenylalanine); 80295-54-1 (Complement C5a)

CHEMICAL NAME:

CAS REGISTRY NO.:

0 (Caseins); 0 (Chemotactic Factors); 0 (Complement C5); 0

(Phytohemagglutinins); 0 (Receptors, Antigen, B-Cell)

L221 ANSWER 10 OF 24 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2006092279 EMBASE

TITLE: Silymarin and silibinin cause G1 and G2-M cell cycle arrest

via distinct circuitries in human prostate cancer PC3

cells: A comparison of flavanone silibinin with

flavanolignan mixture silymarin.

AUTHOR: Deep G.; Singh R.P.; Agarwal C.; Kroll D.J.; Agarwal R.

CORPORATE SOURCE: Prof. R. Agarwal, Department of Pharmaceutical Sciences,

School of Pharmacy, University of Colorado Health Sciences Center, 4200 East Ninth Avenue, Denver, CO 80262, United

States. Rajesh.Agarwal@UCHSC.edu

SOURCE: Oncogene, (16 Feb 2006) Vol. 25, No. 7, pp. 1053-1069.

Refs: 85

ISSN: 0950-9232 E-ISSN: 1476-5594 CODEN: ONCNES

COUNTRY: United Kingdom DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 016 Cancer

028 Urology and Nephrology

030 Pharmacology

037 Drug Literature Index

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 16 Mar 2006

Last Updated on STN: 16 Mar 2006

ABSTRACT: Here, we assessed and compared the anticancer efficacy and associated mechanisms of silymarin and silibinin in human prostate cancer (PCA) PC3 cells; silymarin is comprised of silibinin and its other stereoisomers, including isosilybin A, isosilybin B, silydianin, silychristin and isosilychristin. Silymarin and silibinin (50-100 $\mu g/ml$) inhibited cell proliferation, induced cell death, and caused G1 and G2-M cell cycle arrest in a dose/time-dependent manner. Molecular studies showed that G1 arrest was associated with a decrease in cyclin D1, cyclin D3, cyclin E, cyclin-dependent kinase (CDK) 4, CDK6 and CDK2 protein levels, and CDK2 and CDK4 kinase activity, together with an increase in CDK inhibitors (CDKIs) Kip1/p27 and Cip1/p21. Further, both agents caused cytoplasmic sequestration of cyclin D1 and CDK2, contributing to G1 arrest. The G2-M arrest by silibinin and silymarin was associated with decreased levels of cyclin B1, cyclin A, pCdc2 (Tyr15), Cdc2, and an inhibition of Cdc2 kinase activity. Both agents also decreased the levels of Cdc25B and cell division cycle 25C (Cdc25C) phosphatases with an increased phosphorylation of Cdc25C at Ser216 and its translocation from nucleus to the cytoplasm, which was accompanied by an increased binding with $14-3-3\beta$. Both agents also increased checkpoint kinase (Chk)2 phosphorylation at Thr68 and Ser19 sites, which is known to phosphorylate Cdc25C at Ser216 site. Chk2-specific small interfering RNA largely attenuated the silymarin and silibinin-induced G2-M arrest. An increase in the phosphorylation of histone 2AX and ataxia telangiectasia mutated was also observed. These findings indicate that silymarin and silibinin modulate G1 phase cyclins-CDKs-CDKIs for G1 arrest, and the Chk2-Cdc25C-Cdc2/cyclin B1 pathway for G2-M arrest, together with an altered subcellular localization of critical cell cycle regulators. Overall, we observed comparable effects for both silymarin and silibinin at equal concentrations by weight, suggesting that silibinin could be a major cell cycle-inhibitory component in silymarin. However, other silibinin stereoisomers present in silymarin also contribute to its efficacy, and could be of interest for future investigation. . COPYRGT. 2006 Nature Publishing Group All rights reserved.

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CONTROLLED TERM:
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Medical Descriptors: *cell cycle arrest *antineoplastic activity *prostate cancer cell cycle G1 phase cell cycle G2 phase cell cycle M phase cancer cell culture comparative study drug mechanism drug efficacy stereoisomerism concentration response cell proliferation cell death molecular biology protein content protein determination enzyme activity cytoplasm protein localization cellular distribution enzyme inhibition quantitative analysis enzyme phosphorylation intracellular transport cell nucleus protein transport protein protein interaction cell cycle regulation drug effect drug potency human male controlled study human cell article priority journal Drug Descriptors:

CONTROLLED TERM:

*silibinin: CM, drug comparison *silibinin: DV, drug development *silibinin: PD, pharmacology *silylmarin: CM, drug comparison *silylmarin: DV, drug development *silylmarin: PD, pharmacology *flavanoid: CM, drug comparison *flavanoid: DV, drug development *flavanoid: PD, pharmacology *lignan derivative: CM, drug comparison *lignan derivative: DV, drug development *lignan derivative: PD, pharmacology isosilybin A isosilybin B silidianin silicristin isosilychristin cyclin D1: EC, endogenous compound cyclin D3: EC, endogenous compound cyclin E: EC, endogenous compound cyclin dependent kinase 4: EC, endogenous compound cyclin dependent kinase 6: EC, endogenous compound cyclin dependent kinase 2: EC, endogenous compound cyclin dependent kinase inhibitor 1B: EC, endogenous compound

cyclin dependent kinase inhibitor 1: EC, endogenous

compound

cyclin B1: EC, endogenous compound cyclin A: EC, endogenous compound

cell cycle protein: EC, endogenous compound

cyclin dependent kinase 1: EC, endogenous compound

phosphatase: EC, endogenous compound protein 14 3 3: EC, endogenous compound checkpoint kinase 2: EC, endogenous compound

small interfering RNA

histone H2AX: EC, endogenous compound ATM protein: EC, endogenous compound

unclassified drug

CAS REGISTRY NO.: (silibinin) 22888-70-6; (silidianin) 29782-68-1;

(silicristin) 33889-69-9; (cyclin dependent kinase 4) 147014-97-9; (cyclin dependent kinase 2) 141349-86-2; (phosphatase) 9013-05-2; (protein 14 3 3) 136047-16-0;

(checkpoint kinase 2) 244634-79-5

COMPANY NAME: Sigma Aldrich (United States)

L221 ANSWER 11 OF 24 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2005206506 EMBASE

TITLE: Farnesyltransferase inhibitors induce DNA damage via

reactive oxygen species in human cancer cells.

AUTHOR: Pan J.; She M.; Xu Z.-X.; Sun L.; Yeung S.-C.J.

CORPORATE SOURCE: S.-C.J. Yeung, Dept. of Endocr. Neoplasia/Horm. D., Univ.

Texas M.D. Anderson Cancer C., 1515 Holcombe Boulevard, Houston, TX 77030, United States. syeung@mdanderson.org

SOURCE: Cancer Research, (1 May 2005) Vol. 65, No. 9, pp.

3671-3681. .

Refs: 51

ISSN: 0008-5472 CODEN: CNREA8

COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer

037 Drug Literature Index

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 2 Jun 2005

Last Updated on STN: 2 Jun 2005

ABSTRACT: Farnesyltransferase inhibitors (FTIs) possess antitumor activity. Based on recent findings, we hypothesized that FTIs induce reactive oxygen species (ROS) that damage DNA, leading to DNA damage responses. To test this hypothesis, we investigated the effects of FTIs on the generation of ROS, DNA double-strand breaks (DSB), DNA damage responses, and RhoB, and the effects of quenching ROS on these FTI effects. We evaluated four FTIs in human cancer cell lines of different tissue origins. We found that FTIs induced ROS and DSBs. Suppressing expression of the β-subunit of farnesyltransferase with siRNA did not induce ROS, but slightly attenuated the ROS induced by FTIs. N-acetyl-L-cysteine (NAC), but not caspase inhibitors, blocked FTI-induced DSBs, suggesting that the DSBs were caused by ROS and did not result from apoptosis. The DSBs led to DNA damage responses. H2AX became phosphorylated and formed nuclear foci. The DNA-damage-sensing molecules involved were probably ataxia-telangiectasia mutated protein (ATM) and DNA-dependent protein kinase (DNA-PK) but not ATM- and Rad3-related protein

(ATR). Key components of the homologous recombination and nonhomologous end joining repair pathways (DNA-PK, BRCA1, and NBS1) underwent phosphorylation and formed nuclear foci. RhoB, a mediator of the antineoplastic effect of FTIs and a protein inducible by DNA damage, was increased by FTIs. This increase was blocked by NAC. We concluded that FTIs induced oxidative DNA damage by inducing ROS and initiated DNA damage responses, including RhoB induction, and there was a complex relationship among FTIs, farnesyltransferase, ROS, and RhoB. Our data also imply that inhibitors of DNA repair may accentuate the clinical efficacy of FTIs.

CONTROLLED TERM:

Medical Descriptors:
*DNA damage
*cancer cell

oxidative stress
DNA strand breakage

evaluation

homologous recombination antineoplastic activity

protein induction

ataxia telangiectasia

human

controlled study

human cell

article

priority journal

Drug Descriptors:

*protein farnesyltransferase inhibitor: PD, pharmacology

*DNA: EC, endogenous compound

*reactive oxygen metabolite: EC, endogenous compound

manumycin: PD, pharmacology wortmannin: PD, pharmacology caffeine: PD, pharmacology

doxycycline: PD, pharmacology

2 [[2 [[2 [(2 amino 3 mercaptopropyl)amino] 3
methylpentyl]oxy] 1 oxo 3 phenylpropyl]amino] 4
(methylsulfonyl)butanoic acid isopropyl ester: PD,
pharmacology

n [[5 [(2 amino 3 mercaptopropyl)amino][1,1' biphenyl] 2

yl]carbonyl]methionine: PD, pharmacology

acetylcysteine: PD, pharmacology deferoxamine: PD, pharmacology

ATM protein: EC, endogenous compound

DNA dependent protein kinase: EC, endogenous compound

checkpoint kinase Rad3: EC, endogenous compound

BRCA1 protein: EC, endogenous compound protein nbs1: EC, endogenous compound

RhoB guanine nucleotide binding protein: EC, endogenous

compound

unclassified drug

CAS REGISTRY NO.:

(DNA) 9007-49-2; (manumycin) 52665-74-4; (wortmannin) 19545-26-7; (caffeine) 30388-07-9, 58-08-2; (doxycycline) 10592-13-9, 17086-28-1, 564-25-0; (2 [[2 [[2 amino 3

mercaptopropyl)amino] 3 methylpentyl]oxy] 1 oxo 3
phenylpropyl]amino] 4 (methylsulfonyl)butanoic acid
isopropyl ester) 160141-09-3; (n [[5 [(2 amino 3

mercaptopropyl)amino][1,1' biphenyl] 2

yl]carbonyl]methionine) 170006-72-1; (acetylcysteine)

616-91-1; (deferoxamine) 70-51-9

CHEMICAL NAME:

L 744832; Fti 276

EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights L221 ANSWER 12 OF 24 reserved on STN

ACCESSION NUMBER:

2005396236 EMBASE

TITLE:

The DNA damage pathway regulates innate immune system

ligands of the NKG2D receptor.

AUTHOR:

Gasser S.; Orsulic S.; Brown E.J.; Raulet D.H.

CORPORATE SOURCE:

D.H. Raulet, Department of Molecular and Cell Biology, Cancer Research Laboratory, University of California,

Berkeley, CA 94720-3200, United States.

raulet@uclink.berkeley.edu

SOURCE:

Nature, (25 Aug 2005) Vol. 436, No. 7054, pp. 1186-1190. .

Refs: 27

ISSN: 0028-0836 CODEN: NATUAS

COUNTRY:

United Kingdom

DOCUMENT TYPE:

Journal; Article

FILE SEGMENT:

Immunology, Serology and Transplantation 026

Clinical Biochemistry 029

LANGUAGE:

English English

SUMMARY LANGUAGE: ENTRY DATE:

Entered STN: 29 Sep 2005

Last Updated on STN: 29 Sep 2005

ABSTRACT: Some stimulatory receptors of the innate immune system, such as the NKG2D receptor (also called KLRK1) expressed by natural killer cells and activated CD8(+)T cells, recognize self-molecules that are upregulated in diseased cells by poorly understood mechanisms. Here we show that mouse and human NKG2D ligands are upregulated in non-tumour cell lines by genotoxic stress and stalled DNA replication, conditions known to activate a major DNA damage checkpoint pathway initiated by ATM (ataxia

telangiectasia , mutated) or ATR (ATM- and Rad3-related) protein kinases. Ligand upregulation was prevented by pharmacological or genetic inhibition of ATR, ATM or Chk1 (a downstream transducer kinase in the pathway). Furthermore, constitutive ligand expression by a tumour cell line was inhibited by targeting short interfering RNA to ATM, suggesting that ligand expression in established tumour cells, which often harbour genomic irregularities, may be due to chronic activation of the DNA damage response pathway. Thus, the DNA damage response, previously shown to arrest the cell cycle and enhance DNA repair functions, or to trigger apoptosis, may also participate in alerting the immune system to the presence of potentially dangerous cells.

CONTROLLED TERM:

Medical Descriptors:

*DNA damage *immune system immunoregulation regulatory mechanism natural killer cell gene expression

T lymphocyte activation

genotoxicity

stress

DNA replication enzyme inhibition tumor cell line gene targeting mitosis inhibition

DNA repair apoptosis nonhuman mouse

controlled study

animal cell

article

priority journal
Drug Descriptors:

*natural killer cell receptor NKG2D: EC, endogenous

compound ligand

CD8 antigen: EC, endogenous compound

ATM protein
ATR protein
protein kinase
checkpoint kinase 1
small interfering RNA

cas registry No.: (protein kinase) 9026-43-1

L221 ANSWER 13 OF 24 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights

reserved on STN

ACCESSION NUMBER: 2005357623 EMBASE

TITLE:

ATM Polymorphism and hereditary nonpolyposis colorectal

cancer (HNPCC) age of onset (United States).

AUTHOR:

Jones J.S.; Gu X.; Lynch P.M.; Rodriguez-Bigas M.; Amos

C.I.; Frazier M.L.

CORPORATE SOURCE:

Dr. M.L. Frazier, Department of Epidemiology, University of

Texas, M.D. Anderson Cancer Center, 1515 Holcombe

Boulevard, Houston, TX 77030, United States.

mlfrazier@mdanderson.org

SOURCE:

Cancer Causes and Control, (2005) Vol. 16, No. 6, pp.

749-753. Refs: 16

ISSN: 0957-5243 CODEN: CCCNEN

COUNTRY:

Netherlands

DOCUMENT TYPE: FILE SEGMENT:

Journal; Article
016 Cancer

016

Human Genetics

048 Gastroenterology

LANGUAGE:

English

SUMMARY LANGUAGE:

English

ENTRY DATE:

Entered STN: 9 Sep 2005

Last Updated on STN: 9 Sep 2005

ABSTRACT: Objective: We examined a G-to-A single nucleotide polymorphism of the ATM gene, to determine if it influences hereditary non-polyposis colorectal cancer (HNPCC) age of onset. HNPCC is caused by mutations in mismatch repair genes, especially hMLH1 and hMSH2. ATM germline mutations have been associated with breast and digestive cancers. In a smaller European study, the G-to-A polymorphism was associated with an increased risk of developing an HNPCC-related cancer within HNPCC families. Materials and methods: We genotyped 109 mismatch repair gene (MMR) mutation carriers from 53 HNPCC families for the ATM polymorphism using PCR and single strand conformational polymorphism (SSCP) analysis. We tested the association between the ATM genotypes and HNPCC age of onset by survival analysis. Results: The ATM polymorphism did not significantly modify HNPCC age of onset, nor overall risk, in our population. Conclusions: Although a modifier effect was not seen in our study, future studies that examine the polymorphism in combination with other genetic and environmental factors may elucidate an association. Revealing such associations in MMR mutation carriers may improve risk estimates and help to identify individuals who are genetically susceptible to developing HNPCC at an earlier age. .COPYRGT. Springer 2005.

CONTROLLED TERM:

Medical Descriptors:
*genetic polymorphism
*colorectal cancer

```
onset age
United States
genotype
mismatch repair
gene mutation
family history
polymerase chain reaction
single strand conformation polymorphism
cancer survival
statistical significance
cancer risk
statistical model
genotype environment interaction
risk assessment
genetic susceptibility
  ataxia telangiectasia
human
male
female
major clinical study
controlled study
aged
adult
article
priority journal
Drug Descriptors:
borinic acid derivative
  edetic acid
```

(edetic acid) 150-43-6, 60-00-4 CAS REGISTRY NO.:

EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights L221 ANSWER 14 OF 24 reserved on STN

ACCESSION NUMBER:

2004397797 EMBASE

TITLE: AUTHOR:

The genetics of hypogammaglobulinemia. Grimbacher B.; Schaffer A.A.; Peter H.-H.

CORPORATE SOURCE:

Dr. B. Grimbacher, Div. of Rheumatol./Clin. Immunology, Medical School, University of Freiburg, Hugstetterstrasse 55, 79106 Freiburg, Germany. grimbacher@medizin.ukl.uni-

freiburg.de

SOURCE:

Current Allergy and Asthma Reports, (2004) Vol. 4, No. 5,

pp. 349-358. .

Refs: 90

ISSN: 1529-7322 CODEN: CAARC

COUNTRY:

United Kingdom

DOCUMENT TYPE:

FILE SEGMENT:

Journal; General Review General Pathology and Pathological Anatomy 005

022 Human Genetics

Immunology, Serology and Transplantation 026

037 Drug Literature Index Adverse Reactions Titles 038

LANGUAGE: SUMMARY LANGUAGE: English English

ENTRY DATE:

Entered STN: 30 Sep 2004

Last Updated on STN: 30 Sep 2004

ABSTRACT: Etiologies for human hypogammaglobulinemias are diverse and include genetic and nongenetic causes. Although recent reviews focus on the complex genetics of common variable immunodeficiency, in this review, we survey different causes of hypogammaglobulinemias and discuss possible mechanisms. Copyright .COPYRGT. 2004 by Current Science Inc.

```
CONTROLLED TERM:
                    Medical Descriptors:
                    *molecular genetics
                    *hypogammaglobulinemia: DT, drug therapy
                    *hypogammaglobulinemia: ET, etiology
                    *hypogammaglobulinemia: SI, side effect
                    genetic susceptibility
                    disease classification
                    humoral immune deficiency: DT, drug therapy
                    humoral immune deficiency: ET, etiology
                    humoral immune deficiency: SI, side effect
                    common variable immunodeficiency: ET, etiology
                    immunoglobulin G deficiency: DT, drug therapy
                    immunoglobulin G deficiency: ET, etiology
                    immunoglobulin G deficiency: SI, side effect
                    immunoglobulin A deficiency: ET, etiology
                    immunoglobulin A deficiency: SI, side effect
                    infant disease: ET, etiology
                    linkage analysis
                    Wiskott Aldrich syndrome: DT, drug therapy
                      ataxia telangiectasia: DT, drug therapy
                    DiGeorge syndrome: DT, drug therapy
                    human
                    review
                    Drug Descriptors:
                    immunoglobulin G: EC, endogenous compound
                    immunoglobulin A: EC, endogenous compound
                    cytotoxic agent: AE, adverse drug reaction
                    B lymphocyte antibody: AE, adverse drug reaction
                    gold: AE, adverse drug reaction
                    corticosteroid: AE, adverse drug reaction
                    salazosulfapyridine: AE, adverse drug reaction
                    chloroquine: AE, adverse drug reaction
                      penicillamine: AE, adverse drug reaction
                    hydantoin: AE, adverse drug reaction
                    carbamazepine: AE, adverse drug reaction
                    zonisamide: AE, adverse drug reaction
                    valproic acid: AE, adverse drug reaction
                    captopril: AE, adverse drug reaction
                    fenclofenac: AE, adverse drug reaction
                    polysaccharide vaccine: DT, drug therapy
CAS REGISTRY NO.:
                    (immunoglobulin G) 97794-27-9; (gold) 7440-57-5;
                    (salazosulfapyridine) 599-79-1; (chloroquine) 132-73-0,
                    3545-67-3, 50-63-5, 54-05-7; (penicillamine)
                    2219-30-9, 52-67-5; (hydantoin) 461-72-3;
                    (carbamazepine) 298-46-4, 8047-84-5; (zonisamide)
                    68291-97-4; (valproic acid) 1069-66-5, 99-66-1; (captopril)
                    62571-86-2; (fenclofenac) 34645-84-6
L221 ANSWER 15 OF 24
                     EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:
                    81251649 EMBASE
DOCUMENT NUMBER:
                    1981251649
TITLE:
                    The ataxia telangiectasia clastogenic
                    factor is a low molecular weight peptide.
AUTHOR:
                    Shaham M.; Becker Y.
CORPORATE SOURCE:
                    Dept. Hum. Genet. Molec. Virol., Hadassah-Hebrew Univ. Med.
                    Cent., Jerusalem, Israel
SOURCE:
                    Human Genetics, (1981) Vol. 58, No. 4, pp. 422-424. .
```

CODEN: HUGEDQ

Germany

COUNTRY:

DOCUMENT TYPE:

Journal

FILE SEGMENT:

022

Human Genetics

800

Neurology and Neurosurgery

037

Drug Literature Index

LANGUAGE:

English

ENTRY DATE:

Entered STN: 9 Dec 1991

Last Updated on STN: 9 Dec 1991

ABSTRACT: The clastogenic factor in the plasma of ataxia

(AT) patients and in conditioned medium from AT skin ***telangiectasia*** fibroblast cultures is a peptide with a molecular weight in the range of 500 to 1000. No clastogenic activity could be demonstrated in extracts of cultured AT fibroblasts.

CONTROLLED TERM:

Medical Descriptors:

*ataxia telangiectasia

*chromosome breakage

*clastogenesis *fibroblast cell culture

fibroblast culture molecular weight peptide analysis

serum

in vitro study Drug Descriptors:

*peptide

*phytohemagglutinin

edetic acid

trypsin

CAS REGISTRY NO .:

(phytohemagglutinin) 9008-97-3; (edetic

acid) 150-43-6, 60-00-4; (trypsin)

9002-07-7

COMPANY NAME:

Wellcome103 (United Kingdom); 101; 111; 912; 923; 924

EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights L221 ANSWER 16 OF 24

reserved on STN

ACCESSION NUMBER: 81125089 EMBASE

DOCUMENT NUMBER:

1981125089

TITLE:

Effect of the flavonoid (+) cyanidanol-3 on procollagen

biosynthesis and transport in normal and ataxia

telangiectasia cultured skin fibroblasts.

Becker Y.; Stevely W.; Hamburger Y.; et al. AUTHOR:

Dept. Molec. Virol., Hebrew Univ. Hadassah Med. Sch., CORPORATE SOURCE:

Jerusalem, Israel

SOURCE:

Connective Tissue Research, (1981) Vol. 8, No. 2, pp.

77-84.

CODEN: CVTRBC

COUNTRY:

United States

DOCUMENT TYPE:

Journal

FILE SEGMENT:

Clinical Biochemistry 029

Human Genetics 022

Neurology and Neurosurgery 800 037 Drug Literature Index

LANGUAGE: English

ENTRY DATE:

Entered STN: 9 Dec 1991

Last Updated on STN: 9 Dec 1991

ABSTRACT: The synthesis and secretion of procollagen into the medium of cultures of human skin fibroblasts from normal individuals and from patients

with the genetic disorder, ataxia telangiectasia, are

markedly inhibited by the flavonoid (+) cyanidanol-3. Those proteins which were

11.

secreted into the medium in the presence of cyanidanol were resistant to collagenase treatment (noncollagenous proteins). Polyacrylamide gel electrophoresis revealed the presence of only one noncollagenous protein of 66,000 daltons in the medium of cyanidanol-treated cells as compared with the nine other polypeptides found in the medium of untreated cells.

CONTROLLED TERM: Medical Descriptors:

*ataxia telangiectasia

*fibroblast cell culture cysteine s 35 fibroblast culture

skin

central nervous system

in vitro study human cell

peripheral vascular system reticuloendothelial system

heredity

major clinical study
Drug Descriptors:
*2,2' bipyridine
*aminoacetonitrile
*3 aminopropionitrile

*catechin
*penicillamine
*procollagen
radioisotope

CAS REGISTRY NO.:

(2,2' bipyridine) 366-18-7; (aminoacetonitrile) 151-63-3,

540-61-4; (3 aminopropionitrile) 151-18-8; (catechin)

13392-26-2, 154-23-4; (penicillamine) 2219-30-9,

52-67-5

COMPANY NAME: Zyma (Switzerland); Aldrich (United States)

L221 ANSWER 17 OF 24 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on

STN

ACCESSION NUMBER: 2006:182247 BIOSIS DOCUMENT NUMBER: PREV200600184359

TITLE:

CML progenitor cells have chromsomal instability and display increased DNA damage at DNA fragile sites.

AUTHOR (S):

Dierov, Jamil K. [Reprint Author]; Schoppy, David W.;

Carroll, Martin

CORPORATE SOURCE:

Univ Penn, Philadelphia, PA 19104 USA

SOURCE:

Blood, (NOV 16 2005) Vol. 106, No. 11, Part 1, pp. 563A.

Meeting Info.: 47th Annual Meeting of the

American-Society-of-Hematology. Atlanta, GA, USA. December

10 -13, 2005. Amer Soc Hematol. CODEN: BLOOAW. ISSN: 0006-4971.

DOCUMENT TYPE:

Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE:

English

ENTRY DATE:

Entered STN: 15 Mar 2006

Last Updated on STN: 15 Mar 2006

ABSTRACT: Chronic myelogeneous leukemia (CML) is a two stage disease which progresses to blast crisis over a period of 3-5 years in untreated patients. The BCR/ABL oncogene induces the hyperproliferation associated with chronic phase CML but whether BCR/ABL induces chromosomal instability leading to blast crisis has been controversial. We have previously demonstrated that BCR/ABL delays the repair of DNA double strand breaks and increases chromosomal instability in a murine cell line. Furthermore, we have demonstrated in cell

lines that BCP/ABL disrupts the function of the DNA damage sensing protein, ataxia telangiectasia and rad 3 related (ATR). One of the functions of ATR is to maintain the stability of DNA fragile sites, late replicating sites in the chromosome that are frequently involved in translocations. To determine if BCR/ABL affects the stability of DNA fragile sites in Bat F3 cells that do or do not express BCR/ABL, cells were incubated in low dose aphidicolin for 24 hours to induce fragile site breakage. BCR/ABL expressing cells, but not control cells, demonstrated fragile site damage consistent with a disruption of ATR function in BCR/ABL expressing cells. order to determine if primary patient cells display a genomic instability phenotype, we have analyzed the response to DNA damage in CD34+ cells from normal volunteers and from CIVIL patients seen at the University of Pennsylvania Cancer Center. We first examined the DNA repair response by treating cells for two hours with etoposide. Both normal cells and CML progenitor cells demonstrate DNA double strand breaks as measured by the comet assay, a quantitative assay for DNA double strand breaks. However, in Ph+ cells from the patient sample there was a delay in the repair of DNA double strand breaks as indicated by a significant increase in the olive tail moment at 2 hours and 24 hours after treatment with etoposide. In addition, we analyzed the effect of a two hour exposure to etoposide on chromosome stability as measured by spectral karyotyping (SKY). Normal CD34+ cells and CD34+ cells from patients were treated with etoposide and then allowed to recover for 48 hours before analysis of metaphase spreads. Normal cells demonstrated no spontaneous DNA damage and, after etoposide treatment and repair, demonstrated only modest levels of DNA damage (2 translocations and 5 numerical alterations per 14 metaphases analyzed). In contrast, Ph+ cells demonstrated spontaneous DNA damage in these cell conditions. Furthermore, after etoposide treatment Ph+ cells demonstrated high levels of DNA damage with 9 translocations and 12 numerical alterations in 13 metaphases. These results suggest that Ph+ progenitor cells from patients with CML demonstrate chromosomal instability and suggest a mechanism for progression from CML chronic phase to blast crisis. Full analysis of additional patient samples will be presented. Taken together, we propose that BCR/ABL disrupts ATR function in cell lines and primary cells leading to an increase in chromosomal instability that leads to CML blast crisis.

CONCEPT CODE:

INDEX TERMS:

General biology - Symposia, transactions and proceedings 00520

Cytology - Animal 02506 02508

Cytology - Human Genetics - General 03502

Genetics - Animal 03506

Genetics - Human 03508

Biochemistry studies - General 10060 Biochemistry studies - Nucleic acids, purines and

pyrimidines 10062

Pathology - Therapy 12512

Blood - Blood and lymph studies

Blood - Blood cell studies 15004

Blood - Blood, lymphatic and reticuloendothelial

pathologies 15006

Pharmacology - Clinical pharmacology

Pharmacology - Blood and hematopoietic agents 22008

Neoplasms - Immunology 24003

Neoplasms - Pathology, clinical aspects and systemic

effects 24004

Neoplasms - Therapeutic agents and therapy

Neoplasms - Blood and reticuloendothelial neoplasms 24010

Immunology - General and methods 34502

Immunology - Immunopathology, tissue immunology

Major Concepts

Molecular Genetics (Biochemistry and Molecular Biophysics); Oncology (Human Medicine, Medical

Sciences); Hematology (Human Medicine, Medical Sciences)

INDEX TERMS:

Parts, Structures, & Systems of Organisms

progenitor cell: blood and lymphatics; CD34-positive cell: immune system, blood and lymphatics; chromosome

INDEX TERMS:

chronic myelogeneous leukemia: neoplastic disease, blood

and lymphatic disease, drug therapy, CML

INDEX TERMS:

Chemicals & Biochemicals

DNA; etoposide: antineoplastic-drug, hematologic-drug

INDEX TERMS:

Methods & Equipment

spectral karyotyping: laboratory techniques, genetic

techniques

ORGANISM:

Classifier

Hominidae 86215

Super Taxa

Primates; Mammalia; Vertebrata; Chordata; Animalia

Organism Name human (common)

Taxa Notes

Animals, Chordates, Humans, Mammals, Primates,

Vertebrates

ORGANISM:

Classifier

Muridae 86375

Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Organism Name

Ba/F3 cell line (cell line): murine pro-B cell

Taxa Notes

Animals, Chordates, Mammals, Nonhuman Vertebrates,

Nonhuman Mammals, Rodents, Vertebrates

REGISTRY NUMBER:

GENE NAME:

33419-42-0 (etoposide)

human BCR/ABL gene (Hominidae)

L221 ANSWER 18 OF 24 USPATFULL on STN

ACCESSION NUMBER:

2006:9627 USPATFULL

TITLE:

Shiga toxin B-subunit as a vector for tumor diagnosis

and drug delivery to Gb3 expressing tumors

INVENTOR (S):

Johannes, Ludger, Courbevoie, FRANCE Grierson, David, Versailles, FRANCE Robine, Sylvie, Vanves, FRANCE

Florent, Jean-Claude, Gif-Sur-Yvette, FRANCE Maillard, Philipe, Saint-Cyr-L'Ecole, FRANCE

Roger, Jacky, Villecresnes, FRANCE

PATENT ASSIGNEE(S):

INSTITUT CURIE, Paris Cedex 05, FRANCE (non-U.S.

corporation)

CENTRE NATIONAL DE LA RECHERCHE SCIENTIFIQUE, Paris

Cedex, FRANCE (non-U.S. corporation)

INSTITUT NATIONAL DE LA SANTE ET DE LA RECHERCHE

MEDICALE, Paris Cedex 13, FRANCE (non-U.S. corporation) UNIVERSITE PIERRE ET MARIE CURIE (PARIS VI), Paris,

FRANCE (non-U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION:

US 2006008475

20060112 Α1

APPLICATION INFO.: US 2005-46786

A1 20050201 (11)

RELATED APPLN. INFO.: Continuation of Ser. No. WO 2003-EP9308, filed on 31

Jul 2003, UNKNOWN

NUMBER

DATE

PRIORITY INFORMATION:

EP 2002-291962

20020802

DOCUMENT TYPE:

Utility

FILE SEGMENT:

APPLICATION

LEGAL REPRESENTATIVE:

BIRCH STEWART KOLASCH & BIRCH, PO BOX 747, FALLS

CHURCH, VA, 22040-0747, US

NUMBER OF CLAIMS:

1

.

EXEMPLARY CLAIM: NUMBER OF DRAWINGS:

16 Drawing Page(s)

LINE COUNT:

1412

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

The invention relates to new compounds for cancer therapy or diagnosis and to the use of a non-toxic B subunit of Shiga toxin mutant as a vector for diagnostic products or drugs in over-expressing Gb3 receptor cells, such compounds having the following formula: STxB-Z(n)-Cys-Y(m)-T wherein-STxB is the Shiga Toxin B subunit or a functional equivalent thereof, --Z(n) wherein n is 0 or 1, Z is an amino-acid residue devoid of sulfydryl groups, or is a polypeptide, --T is a molecule linked by a covalent bound to the S part of Cys, selected from: agents for in vivo diagnosis, cytotoxic agents, prodrugs, or enzymes for the conversion of a prodrug to a drug, --Y(m) wherein m is 0 or 1, Y is a linker between T and Cys, which is either cleavable or not cleavable for the release of T after the internalization of the hybrid compound into cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD Babilon, R. W., K. J. Soprano, and E. E. Henderson. 1985. Hypersensitivity and reduced inhibition of DNA synthesis in ataxia telangiectasia lymphoblasts treated with low levels of neocarzinostatin. Mutat. Res. 146:79-87.

DETD Shiloh, Y., E. Tabor, and Y. Becker. 1982. Cellular hypersensitivity to neocarzinostatin in ataxia-telangiectasia skin fibroblasts. Cancer Res. 42:2247-2249.

IT 67-43-6D, Diethylenetriaminepentaacetic acid, complex with gadolinium and ethoxybenzyl 7440-54-2D, Gadolinium, complex with acetic acid derivs. 7440-54-2D, Gadolinium, polymer complexes 83678-67-5, Gd-DoTA 651740-21-5

(Shiga toxin B-subunit as a vector for tumor diagnosis and drug delivery to Gb3-expressing tumors)

IT 67-43-6D, Diethylenetriaminepentaacetic acid, complex with qadolinium and ethoxybenzyl

(Shiga toxin B-subunit as a vector for tumor diagnosis and drug delivery to Gb3-expressing tumors)

RN 67-43-6 USPATFULL

L221 ANSWER 19 OF 24 USPATFULL on STN

ACCESSION NUMBER: 2005:292956 USPATFULL

Method for determination and quantification of TITLE:

radiation or genotoxin exposure

D'Andrea, Alan D., Winchester, MA, UNITED STATES INVENTOR(S):

Dana Farber Cancer Center (U.S. corporation) PATENT ASSIGNEE(S):

> NUMBER KIND DATE -----_____

US 2005255502 A1
US 2005-46346 A1 PATENT INFORMATION: 20051117

APPLICATION INFO.: 20050128 (11)

> NUMBER DATE _____

PRIORITY INFORMATION: US 2004-540380P 20040130 (60)

DOCUMENT TYPE: Utility APPLICATION FILE SEGMENT:

LEGAL REPRESENTATIVE: PALMER & DODGE, LLP, KATHLEEN M. WILLIAMS, 111

HUNTINGTON AVENUE, BOSTON, MA, 02199, US

NUMBER OF CLAIMS: EXEMPLARY CLAIM:

NUMBER OF DRAWINGS: 14 Drawing Page(s)

2700 LINE COUNT:

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

The present invention discloses methods for detecting exposure of a living subject to genotoxic agents, testing sensitivity to a genotoxic agent, and determining DNA damage caused by exposure to an agent, comprising detecting the presence of FANCD2-containing foci from a sample collected from said subject. The presence of concentrated foci is indicative of DNA damage, and the degree of foci formation is correlated with degree of exposure. Diagnostic reagents contain a ligand that binds to human FANCD2 associated with a detectable label. Kits for detecting DNA damage in a biological sample contain such diagnostic reagents and signal detection components. The invention further discloses methods for identifying agents which modulate the ability of FANCD2-containing foci to form. Among other things, such agents are potentially useful chemosensitizing agents or may confer protection against damage caused by genotoxic agents.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

. . Kim, S. T., Lane, W. S., Kastan, M. B., and D'Andrea, A. D. DETD

(2002). Convergence of the Fanconi anemia and ataxia telangiectasia signaling pathways. Cell 109, 459-472.

Wu, X., Petrini, J. H., Heine, W. F., Weaver, D. T., Livingston, D. M.,. . .

IT 70-51-9, Desferrioxamine (FA/BRCA pathway agonist; antibodies to monoubiquitinated FANCD2 protein and method for determination and quantification of radiation or genotoxin exposure)

TΤ 70-51-9, Desferrioxamine

(FA/BRCA pathway agonist; antibodies to monoubiquitinated FANCD2 protein and method for determination and quantification of radiation or genotoxin exposure)

70-51-9 USPATFULL RN

Butanediamide, N'-[5-[[4-[[5-(acetylhydroxyamino)pentyl]amino]-1,4-CN dioxobutyl]hydroxyamino]pentyl]-N-(5-aminopentyl)-N-hydroxy- (9CI) (CA INDEX NAME)

PAGE 1-B

-(CH₂)₅-NH₂

L221 ANSWER 20 OF 24 USPATFULL on STN

ACCESSION NUMBER:

2005:286471 USPATFULL

TITLE:

Methods of treating ankylosing spondylitis using

anti-TNF antibodies and peptides of human tumor

necrosis factor

INVENTOR(S):

Le, Junming, Forest Hills, NY, UNITED STATES Vilcek, Jan T., Manhattan, NY, UNITED STATES Daddona, Peter E., Menlo Park, CA, UNITED STATES Ghrayeb, John, Downingtown, PA, UNITED STATES Knight, David M., Berwyn, PA, UNITED STATES Siegel, Scott A., Ringoes, NJ, UNITED STATES Shealy, David J., Downingtown, PA, UNITED STATES Centocor, Inc., Malvern, PA, UNITED STATES (U.S.

PATENT ASSIGNEE(S):

corporation)

New York University, New York, NY, UNITED STATES (U.S.

corporation)

NUMBER KIND DATE _____

PATENT INFORMATION: APPLICATION INFO .:

US 2005249735 A1 US 2004-10954 A1 20051110

RELATED APPLN. INFO.:

20041213 (11) Continuation-in-part of Ser. No. US 2003-637759, filed

on 8 Aug 2003, PENDING Continuation-in-part of Ser. No.

US 2001-927703, filed on 10 Aug 2001, PENDING

Continuation of Ser. No. US 2001-756398, filed on 8 Jan 2001, GRANTED, Pat. No. US 6835823 Continuation-in-part

of Ser. No. US 2001-920137, filed on 1 Aug 2001,

PENDING

NUMBER DATE

PRIORITY INFORMATION:

----20000807 (60)

US 2000-223360P US 2000-236826P

20000929 (60)

DOCUMENT TYPE:

Utility

FILE SEGMENT:

APPLICATION

LEGAL REPRESENTATIVE:

HAMILTON, BROOK, SMITH & REYNOLDS, P.C., 530 VIRGINIA

ROAD, P.O. BOX 9133, CONCORD, MA, 01742-9133, US

NUMBER OF CLAIMS: EXEMPLARY CLAIM:

NUMBER OF DRAWINGS:

37 Drawing Page(s)

LINE COUNT:

7263

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

Anti-TNF antibodies, fragments and regions thereof which are specific

for human tumor necrosis factor- α (TNF α) and are useful in vivo diagnosis and therapy of a number of TNF α -mediated pathologies and conditions, including ankylosing spondylitis, as well as polynucleotides coding for murine and chimeric antibodies, methods of producing the antibody, methods of use of the anti-TNF antibody, or fragment, region or derivative thereof, in immunoassays and immunotherapeutic approaches are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . ataxia, Friedreich's ataxia, cerebellar cortical degenerations, multiple systems degenerations (Mencel, Dejerine-Thomas, Shi-Drager, and MachadoJoseph)); and systemic disorders (Refsum's disease, abetalipoprotemia, ataxia, telangiectasia, and mitochondrial multi-system disorder); demyelinating core disorders, such as multiple sclerosis, acute transverse myelitis; disorders of the motor unit, such. . .

DETD . . . spinal ataxia, Friedreich's ataxia, cerebellar cortical degenerations, multiple systems degenerations (Mencel, Dejerine-Thomas, Shi-Drager, and Machado-Joseph); systemic disorders (Refsum's disease, abetalipoprotemia, ataxia, telangiectasia, and mitochondrial multi.system disorder); demyelinating core disorders, such as multiple sclerosis, acute transverse myelitis; and disorders of the motor unit'. . .

· IT 50-07-7, Mitomycin C 50-78-2, Aspirin 51-43-4, Epinephrine 52-28-8, Codeine phosphate 52-67-5, Penicillamine 53-86-1, Indomethacin 54-05-7, Chloroquine 59-05-2, Methotrexate 89-57-6, 103-90-2, Paracetamol 118-42-3, Hydroxychloroquine 321-64-2, Tac rine 446-86-6, Azathioprine 469-62-5, Dextropropoxyphene 5003-48-5, Benorylate 9002-72-6, Growth hormone 11096-26-7, 15307-86-5, Diclofenac Erythropoietin 12244-57-4, Myocrisin 15687-27-1, Ibuprofen 16110-51-3, Cromolyn 22204-53-1, Naprosyn 23214-92-8, Doxorubicin 28109-92-4, Methylxanthine 34031-32-8, Auranofin 36330-85-5, Fenbufen 41340-25-4, Etodolac 120014-06-4, 121181-53-1, Filgrastim 123774-72-1, Sargramostim Donepezil 143831-71-4, Dornase alfa

(methods of treating ankylosing spondylitis using anti-tumor necrosis factor antibodies and peptides of human tumor necrosis factor)

IT 52-67-5, Penicillamine

(methods of treating ankylosing spondylitis using anti-tumor necrosis factor antibodies and peptides of human tumor necrosis factor)

RN 52-67-5 USPATFULL

CN D-Valine, 3-mercapto- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

L221 ANSWER 21 OF 24 USPATFULL on STN

ACCESSION NUMBER:

2005:144174 USPATFULL

TITLE:

COMPOSITIONS AND METHODS FOR TREATING CELLS HAVING

DOUBLE MINUTE DNA

INVENTOR(S):

WAHL, GEOFFREY M., SAN DIEGO, CA, UNITED STATES

SHIMIZU, NORIAKI, HIROSHIMA, JAPAN

KANDA, TERU, LA JOLLA, CA, UNITED STATES SHEPARD, H. MICHAEL, RANCHO SANTA FE, CA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005123909	A 1	20050609	
	US 6946259	B2	20050920	
APPLICATION INFO.:	US 1999-229229	A1	19990112	(9)

NUMBER DATE

PRIORITY INFORMATION: US 1998-71146P 19980112 (60) US 1998-77644P 19980311 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: MCDERMOTT, WILL & EMERY, 4370 LA JOLLA VILLAGE DRIVE,

SUITE 700, SAN DIEGO, CA, 92122, US

NUMBER OF CLAIMS: 23 EXEMPLARY CLAIM: 1-32

NUMBER OF DRAWINGS: 14 Drawing Page(s)

LINE COUNT: 1977

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

This invention provides methods by which test substances can be screened for their ability to inhibit, enhance or eliminate double minute (DM) or extrachromosomal DNA by micronucleation in cells. This invention also provides a method for inducing maturation or death of a cell having the capacity to generate micronuclei. It also provides a method of treating a disease in a subject, the cells correlated with the disease having DM and extrachromosomal DNA as well as the capacity to generate micronuclei to capture them. Further provided is a method of detecting chromosomal and extrachromosomal DNA in a cell.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . B. Vogelstein and A. J. Fonace. 1992. "A mammalian cell cycle checkpoint pathway utilizing p53 and GADD45 is defective in ataxia-telangiectasia" Cell. 7:587-97.

Kuerbitz, S. J., B. S. Plunkett, W. V. Walsh and M. B. Kastan. 1992.
"Wild-type p53 is. . .

IT 67-68-5, DMSO, biological studies 70-51-9, Deferoxamine

91-64-5, Coumarin 98-92-0, Nicotinamide 1455-77-2, Guanazole

38966-21-1, Aphidicolin 51321-79-0, PALA

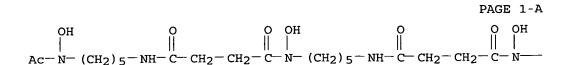
(compns. and methods for identifying therapeutic agents and for treating cells having double minute DNA)

IT 70-51-9, Deferoxamine

(compns. and methods for identifying therapeutic agents and for treating cells having double minute DNA)

RN 70-51-9 USPATFULL

CN Butanediamide, N'-[5-[[4-[[5-(acetylhydroxyamino)pentyl]amino]-1,4-dioxobutyl]hydroxyamino]pentyl]-N-(5-aminopentyl)-N-hydroxy-(9CI) (CAINDEX NAME)



PAGE 1-B

-(CH₂)₅-NH₂

L221 ANSWER 22 OF 24 USPATFULL on STN

ACCESSION NUMBER: 2003:161945 USPATFULL

TITLE: Diagnosis and management of infection caused by

chlamydia

INVENTOR (S): Mitchell, William M., Nashville, TN, United States

Stratton, Charles W., Nashville, TN, United States

PATENT ASSIGNEE(S): Vanderbilt University, Nashville, TN, United States

(U.S. corporation)

	NUMBER	KIND	DATE		
PATENT INFORMATION:	US 6579854	B1	20030617		
APPLICATION INFO.:	US 1998-73661		19980506	(9)	
RELATED APPLN. INFO.:	Continuation-in-	part of	Ser. No.	US 1998-25174, filed	
	on 18 Feb 1998 Continuation-in-part of Ser. No. US				
	1998-25521, filed	d on 18	Feb 1998,	, now abandoned	
	Continuation-in-	part of	Ser. No.	US 1998-25176, filed	
	on 18 Feb 1998, now patented, Pat. No. US 6258532				
	Continuation-in-part of Ser. No. US 1997-911593, filed				
	on 14 Aug 1997, 1	now abar	ndoned		

	NUMBER	DATE				
PRIORITY INFORMATION:	US 1997-45689P	19970506	(60)			
	US 1997-45739P	19970506	(60)			
	US 1997-45779P	19970506	(60)			
	US 1997-45780P	19970506	(60)			
	US 1997-45787P	19970506	(60)			
	US 1996-23921P	19960814	(60)			
DOCUMENT TYPE:	Utility					
FILE SEGMENT:	GRANTED					
PRIMARY EXAMINER:	Weddington, Kevin E.					
LEGAL REPRESENTATIVE:	Clark & Elbing LLP					

NUMBER OF CLAIMS: . 9

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 6 Drawing Figure(s); 4 Drawing Page(s)

LINE COUNT: 4353

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

The present invention provides a unique approach for the diagnosis and management of infections by Chlamydia species, particularly C. pneumoniae. The invention is based, in part, upon the discovery that a combination of agents directed toward the various stages of the chlamydial life cycle is effective in substantially reducing infection. Products comprising combination of antichlamydial agents, novel compositions and pharmaceutical packs are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

. . . Friedreich's ataxia, cerebellar cortical degenerations. multiple systems degenerations (Mencel, Dejerine-Thomas, Shi-Drager, and Machado Joseph)); and systemic disorders (Refsum's disease, abetalipoprotemia, ataxia, telangiectasia, and mitochondrial multi-system disorder); demyelinating core disorders, such

1.5

as multiple sclerosis, acute transverse myelitis; disorders of the motor unit, such.

54-85-3, Isoniazide 55-22-1, 52-67-5, Penicillamine Isonicotinic acid, biological studies 69-53-4, Ampicillin 443-48-1, 8064-90-2, 564-25-0, Doxycycline 6998-60-3, Rifamycin Metronidazole 10118-90-8, Minocycline 13292-46-1, Rifampin 26787-78-0, 81103-11-9, Clarithromycin 36877-68-6, Nitroimidazole Amoxicillin 83905-01-5, Azithromycin 82419-36-1, Ofloxacin

(for Chlamydia infection treatment; diagnosis and management of infection caused by Chlamydia)

52-67-5, Penicillamine IT

(for Chlamydia infection treatment; diagnosis and management of infection caused by Chlamydia)

52-67-5 USPATFULL RN

D-Valine, 3-mercapto- (9CI) (CA INDEX NAME) CN

Absolute stereochemistry.

L221 ANSWER 23 OF 24 WPIX COPYRIGHT 2006 THE THOMSON CORP on STN

ACCESSION NUMBER:

WPIX 2004-449666 [42]

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C2004-168507

Use of agent(s) that elevate intracellular cyclic TITLE: adenosine monophosphate or intracellular calcium levels

in neural tissue for modulating neurogenesis to treat

central nervous system disorder.

DERWENT CLASS:

BERTILSSON, G; ERLANDSSON, R; FRISEN, J; HAEGESTRAND, A; INVENTOR(S):

HAGGBLAD, J; HEIDRICH, J; HELLSTROM, K; JANSSON, K;

KORTESMAA, J; LINDQUIST, P; LUNDH, H; MCGUIRE, J; MERCER,

A; NJBERG, K; OSSOINAK, A; PATRONE, C; RONNHOLM, H;

WIKSTROM, L; ZACHRISSON, O; HAEGGBLAD, J; HELLSTROEM, K; ROENNHOLM, H; WIKSTROEM, L; HAEGERSTRAND, A; HELLSTROM,

N; NYBERG, K; HELLSTROEM, N

(BERT-I) BERTILSSON G; (ERLA-I) ERLANDSSON R; (FRIS-I) PATENT ASSIGNEE(S):

FRISEN J; (HAEG-I) HAEGESTRAND A; (HAGG-I) HAGGBLAD J; (HEID-I) HEIDRICH J; (HELL-I) HELLSTROM K; (JANS-I) JANSSON K; (KORT-I) KORTESMAA J; (LIND-I) LINDQUIST P;

(LUND-I) LUNDH H; (MCGU-I) MCGUIRE J; (MERC-I) MERCER A; (NEUR-N) NEURONOVA AB; (NJBE-I) NJBERG K; (OSSO-I)

OSSOINAK A; (PATR-I) PATRONE C; (RONN-I) RONNHOLM H; (WIKS-I) WIKSTROM L; (ZACH-I) ZACHRISSON O; (HAEG-I)

HAEGGBLAD J; (HELL-I) HELLSTROEM K; (ROEN-I) ROENNHOLM H;

(WIKS-I) WIKSTROEM L

COUNTRY COUNT: 108

PATENT INFORMATION:

PG MAIN IPC LΑ WEEK KIND DATE PATENT NO

WO 2004045592 A2 20040603 (200442)* EN 77 A61K031-00 RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US

UZ VC VN YU ZA ZM ZW

AU 2003280117 A1 20040615 (200470) A61K031-00 US 2005009742 A1 20050113 (200506) A61K038-17 US 2005009847 A1 20050113 (200506) A61K038-17 US 2005209142 A1 20050922 (200563) A61K038-22 EP 1583541 A2 20051012 (200568) EN A61K031-675

R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LT LU LV

MC MK NL PT RO SE SI SK TR

US 6969702 B2 20051129 (200578) A01N037-18 US 2006079448 A1 20060413 (200626) A61K038-22 JP 2006514630 W 20060511 (200635) 70 A61K045-00

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE	
WO 2004045592 AU 2003280117	A2 A1	WO 2003-IB5311 AU 2003-280117	20031120	
US 2005009742	Al Provisional CIP of	US 2002-427912P US 2003-718071 US 2004-850055	20021120 20031120 20040519	
US 2005009847	A1 Provisional	US 2001-030033 US 2002-427912P US 2003-718071	20040313 20021120 20031120	
US 2005209142	Al Provisional CIP of CIP of	US 2002-427912P US 2003-718071 US 2004-850055	20021120 20031120 20040519	
EP 1583541	A2	US 2004-993667 EP 2003-772495 WO 2003-IB5311	20041119 20031120 20031120	
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JP 2006514630	W	WO 2003-IB5311 JP 2004-553032	20031128 20031120 20031120	

FILING DETAILS:

PAT	TENT NO	KII	ND		F	ATENT	NO	
AU	2003280	117 A1	Based	on	WO	200404	1559	2
EP	1583541	A2	Based	on	WO	200404	1559	2
US	20060794	148 A1	Div ex		US	696970)2	
JP	20065146	530 W	Based	on	WO	200404	1559	2
PRIORITY	APPLN.	20	5 2002- 003-718 004-850 004-993	071 055 667	2003 2004 2004	002112 1120; 0519; 1119; 1128	US US	US

INT. PATENT CLASSIF.:

MAIN: A01N037-18; A61K031-00; A61K031-675; A61K038-17;

A61K038-22; A61K045-00

SECONDARY: A61K031-352; A61K031-4015; A61K031-522; A61K031-7042;

A61K031-7076; A61K035-66; A61K035-74; A61K038-00; A61K038-12; A61K038-23; A61P009-00; A61P009-10; A61P019-00; A61P025-00; A61P025-14; A61P025-16;

A61P025-28; A61P037-00; A61P043-00

BASIC ABSTRACT:

WO2004045592 A UPAB: 20040702

NOVELTY - In modulating neurogenesis in neural tissue of a patient exhibiting symptom(s) of central nervous system disorder, such as neurodegenerative, ischemic or learning and memory disorder or neurological trauma, at least one agent (A) that elevates intracellular cyclic adenosine monophosphate (cAMP) levels or at least one agent (B) that elevates intracellular Ca2+ levels in the neural tissue, is administered where (A) modulates and (B) induces neurogenesis.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

- (1) method (M1) for increasing the intracellular levels of cAMP in a cell or method (M2) for stimulating intracellular cAMP in a cell of a patient involving: contacting the cell, or administering to the patient an agent selected from (des-Arg9, Leu8) -bradykinin, (Des-Arg9) -bradykinin, alpha-neoendorphin, CART (61-102), DTLET, eledoisin, urotensin II, (NIe8, Tyr34) -parathyroid hormone (1-34) amide and/or (Cys3,6, Tyr8, Pro9) -Substance P; and
 - (2) modulating neurogenesis in vitro by:
- (a) culturing a population of neural cells, comprising neural stem cells:
- (b) adding at least one neurogenesis modulating agent to the cultured cells; and
- (c) repeating step (a) and (b) to achieve a desired level of neurogenesis.

ACTIVITY - Nootropic; Neuroprotective; CNS-Gen.; Cerebroprotective; Vasotropic; Anticonvulsant; Antiparkinsonian; Hemostatic; Hypertensive; Muscular-Gen.; Ophthalmological; Antiinflammatory; Analgesic; Antidiabetic.

MECHANISM OF ACTION - Neurogenesis modulator; Neural stem or progenitor cell proliferation, differentiation and/or migration modulator; Neural tissue G-protein coupled receptor activator; Neurogenesis inducer; Intracellular neural cAMP enhancer; Intracellular neural cAMP stimulator; Intracellular neural Ca2+ enhancer.

N-6,2-O-Dibutyryl-adenosine (a) was studied for induction of proliferation and increase in the cAMP levels in mouse adult neural stem cells by adding to the culture at 100 nM. After 15 minutes cAMP levels were measured and after 4 days ATP levels were measured. Control cells were treated with vehicle. ATP Level (nM, ATP/well), fold induction of ATP, cAMP level (pmol/well) and fold induction of cAMP in the cells treated with (a)/vehicle were found to be: 13.9 plus or minus 1.1/9.3 plus or minus 0.6, 1.5/1, 0.1 plus or minus 0.01/0.02 plus or minus 0.01 and 4.5/-, respectively.

USE - For modulating neurogenesis in neural tissue of a patient exhibiting at least one symptom of central nervous system disorder, such as neurodegenerative disorder, ischemic disorder, neurological trauma and learning and memory disorder, e.g. Parkinson's disease and Parkinson's disorders, Huntington's disease, Alzheimer's disease, multiple sclerosis, amyotrophic lateral sclerosis, Shy-Drager syndrome, progressive supranuclear palsy, Lewy body disease, spinal ischemia, ischemic stroke, cerebral infarction, spinal cord injury, cancer-related brain and spinal cord injury, multi-infarct dementia and geriatric dementia; for increasing

the intracellular cAMP levels in a cell (preferably a cell from a neural tissue); for stimulating intracellular cAMP in a cell of a patient; and for in vitro modulation of neurogenesis (claimed). Also useful for the treatment of idiopathic orthostatic hypotension, structural lesions of cerebellum (e.g. those associated with infarcts, hemorrhage or tumors), spinocerebellar degenerations (e.g. associated with Friedreich's ataxia), abetalioproteinemia, Refsum's disease, cerebellar ataxia, multiple systems atrophy, ataxia-telangiectasia, mitochondrial multisystem disorders, acute disseminated encephalomyelitis, adrenoleukodystrophy, adrenomyeloneuropathy, Leber's hereditary optic dystrophy, HTLV-associated myelopathy, progressive bulbar palsy, progressive muscular atrophy, primary lateral sclerosis, progressive pseudobulbar palsy, spinal muscular atrophy, plexopathy, acute branchial neuritis, peripheral neuropathy, multiple mononeuropathies, polyneuropathies, nerve palsy, carpal tunnel syndrome, peroneal nerve palsy, radial nerve palsy, Guillain-Barre syndrome, chronic relapsing, hereditary motor and sensory neuropathy, myasthenia gravis, neuro-ophthalmological disorders, cranial nerve palsies, trigeminal neuralgia, Bell's palsy, glossopharyngeal neuralgia, radiation-induced injury of the nervous system, chemotherapy-induced neuropathy, taxol neuropathy, vincristine neuropathy, diabetic neuropathy, autonomic neuropathy, polyneuropathy, mononeuropathy, transient ischemic attacks, subclavian steal syndrome, drop attacks, ischemic stroke, hemorrhagic stroke and brain infarction; for the detection of endogenous agents in cells that are involved in the mediation of signal transduction pathways in the regulation of neurogenesis function; and in the diagnosis of diseases and determine the role of stem and progenitor cells in the disease.

ADVANTAGE - The agent modulates neurogenesis in neural tissue by modulating proliferation, differentiation, migration or survival of neural stem cells or progenitor cells in the tissue; by maintaining or increasing the amount or percentage of doublecortin positive cells in the neural tissue relative to a patient not dosed with the agent or by activation of a G-protein coupled receptor in the neural tissue. The method results in elevation of cAMP levels of the neural stem cells by at least 20% compared to untreated tissue. The in vivo induction of neurogenesis allows treatment of disorders caused by cell loss, injury or disease by endogenous replacement and obviates the need for transplanting foreign cells into a patient. Neurogenesis can also be induced by administration of the neurogenesis-modulating agent directly into a desired site, which avoids unnecessary systemic administration and possible side effects and further provides an alternative to the use of drugs and the controversial use of large quantities of embryonic tissue for treatment of Parkinson's disease.

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Dwg.0/1
FILE SEGMENT:
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FIELD AVAILABILITY:
                      AB; DCN
MANUAL CODES:
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2004-449666 [42]
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DC B02 B03

ICM A01N037-18; A61K031-00; A61K031-675; A61K038-17; A61K038-22; IC A61K045-00 ICS A61K031-352; A61K031-4015; A61K031-522; A61K031-7042; A61K031-7076;

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        M904 M905 M910 P444 P446 P510 P517 P525 P526 P528 P617 P922
        DCN: R00971-K; R00971-T; R00971-U;
             RA0055-K; RA0055-T; RA0055-U
M2 *28* D011 D015 D932 H1 H181 H2 H201 H212 J5 J522 J581 L9 L910
        M210 M211 M213 M231 M262 M273 M281 M282 M314 M321 M332 M342 M381
        M391 M412 M511 M520 M530 M540 M781 M904 M905 P444 P446 P510 P517
        P525 P526 P528 P617 P922
        DCN: R20131-K; R20131-T; R20131-U
    M282 M320 M412 M511 M520 M530 M540 M781 M904 M905 P444 P446 P510
        P517 P525 P526 P528 P617 P922
        DCN: R21071-K; R21071-T; R21071-U
   *30* F012 F014 F015 F522 G013 G100 H5 H594 H9 J5 J521 J581 L9
        L921 M1 M123 M131 M210 M211 M240 M271 M281 M320 M413 M510 M521
        M531 M540 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617
        DCN: R11079-K; R11079-T; R11079-U
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M2 *31* D023 D210 M210 M211 M214 M232 M240 M282 M320 M412 M511 M520 M530
        M540 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
        DCN: RAAWOS-K; RAAWOS-T; RAAWOS-U
M2 *32* D011 D023 D631 G015 G100 H5 H543 H8
                                                    M123 M132 M210 M211
                                              M1
        M272 M283 M311 M321 M342 M412 M511 M520 M531 M540 M640 M781 M904
        M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
        DCN: R04935-K; R04935-T; R04935-U
M2 *33* C017 C100 C720 C800 C801 C803 C804 C805 C806 C807 F011 F013 F522
        G013 G015 G019 G100 H1 H181 H2 H201 H5
                                                    H581 H6
                                                               H602 H609
                       L7 L721 M1 M121 M129 M132 M139 M150 M280 M311
        H643 H8
                  K0
        M312 M321 M322 M332 M342 M343 M373 M393 M411 M510 M521 M533 M540
        M640 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
        DCN: RA7PW6-K; RA7PW6-T; RA7PW6-U
   *34* F013 F014 F016 F530 G013 G100 J5
                                           J521 K0
                                                          L110 L2
                                                     L1
             L941 M1 M113 M210 M211 M240 M273 M281 M320 M413 M510 M521
        M531 M540 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617
        DCN: RA06CF-K; RA06CF-T; RA06CF-U
                       H101 H182 H2
M2
    *35* D015 D931 H1
                                     H212 J5
                                                J522 L9
                                                          L910 M210 M211
        M273 M282 M312 M321 M332 M342 M383 M391 M412 M511 M520 M530 M540
        M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
        DCN: R03933-K; R03933-T; R03933-U
   *36* G015 G019 G100 H4 H404 H444 H7
                                           H721 H8
M2
                                                     J5
                                                          J581 M1
        M135 M280 M312 M321 M332 M342 M372 M391 M414 M510 M520 M532 M540
        M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
        DCN: R19932-K; R19932-T; R19932-U
   *37* C017 C100 C800 C801 C803 C804 C805 C806 C807 D011 D013 D830 H2
М2
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        H211 J0
        M273 M281 M282 M320 M411 M511 M520 M530 M540 M640 M781 M904 M905
        P444 P446 P510 P517 P525 P526 P528 P617 P922
        RIN: 01190
        DCN: R11856-K; R11856-T; R11856-U
   *38* C017 C100 C800 C801 C803 C804 C805 C806 C807 D011 D022 E800 F011
M2
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        M313 M321 M332 M342 M344 M353 M383 M391 M411 M511 M521 M530 M540
        M640 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
        DCN: RA6KYT-K; RA6KYT-T; RA6KYT-U
M2 *39* F012 F013 F014 F015 F016 F431 F432 J5
                                                J521 K0
                                                          L1
                  M116 M210 M211 M240 M281 M320 M413 M510 M522 M530 M540
        L941 M1
        M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
        DCN: R11076-K; R11076-T; R11076-U
M2 *40* D013 D016 D023 D026 D029 D030 D220 H4
                                                H403 H421 H462 H7
                               J261 J5 J521 M210 M211 M212 M240 M262
        H721 H8
                  JO
                       J011 J2
        M281 M283 M320 M412 M511 M520 M530 M540 M781 M800 M904 M905 P444
        P446 P510 P517 P525 P526 P528 P617 P922
        RIN: 03577
        DCN: R04356-K; R04356-T; R04356-U
M2 *41* B615 B701 B713 B720 B815 B831 B840 D011 D012 D013 D016 D019 D030
                                    H201 J0 J012 J2 J222 L910 M210
                       H100 H122 H2
        D160 D931 H1
        M213 M231 M262 M282 M320 M411 M512 M520 M530 M540 M781 M904 M905
        P444 P446 P510 P517 P525 P526 P528 P617 P922
        RIN: 08000
        DCN: RADLVX-K; RADLVX-T; RADLVX-U
   *42* B615 B701 B713 B720 B815 B831 B840 D011 D012 D013 D016 D019 D160
                  H100 H122 H2 H201 H4 H401 H421 H6 H602 H621 H8
        D931 H1
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        P510 P517 P525 P526 P528 P617 P922
        RIN: 08000 08000
        DCN: R18084-K; R18084-T; R18084-U; R18085-K; R18085-T; R18085-U
M2 *43* B615 B701 B713 B720 B815 B831 B840 D011 D012 D013 D016 D019 D160
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        P510 P517 P525 P526 P528 P617 P922
        RIN: 08000
        DCN: RA04NE-K; RA04NE-T; RA04NE-U
M2 *44* D011 D024 D640 G013 G100 H403 H443 H602 H641 K431 K432 M113 M210
        M211 M271 M280 M281 M320 M412 M510 M511 M520 M530 M531 M540 M620
        M650 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
        RIN: 01843
        DCN: RAEJ8B-K; RAEJ8B-T; RAEJ8B-U
M2 *45* C017 C100 C800 C801 C803 C804 C805 C806 C807 G015 G100 H1 H100
        M510 M520 M531 M540 M640 M781 M904 M905 P444 P446 P510 P517 P525
        P526 P528 P617 P922
        DCN: R11187-K; R11187-T; R11187-U
M2 *46* C017 C100 C720 C800 C801 C803 C804 C805 C806 C807 D011 D023 D030
        E310 H1 H181 H2 H201 H4 H402 H442 H8 M210 M211 M273 M280
        M281 M320 M411 M511 M520 M530 M540 M640 M781 M904 M905 P444 P446
        P510 P517 P525 P526 P528 P617 P922
        RIN: 05171
        DCN: R22680-K; R22680-T; R22680-U
   *47* B215 B713 B720 B819 B831 C108 C720 C800 C801 C802 C803 C804 C805
        C807 F014 F521 H1 H100 H181 M280 M312 M321 M332 M342 M373 M391
        M411 M510 M521 M530 M540 M640 M781 M904 M905 P444 P446 P510 P517
        P525 P526 P528 P617 P922
        DCN: R12275-K; R12275-T; R12275-U
M2 *48* C316 D011 D022 D601 H103 H181 J012 J172 K353 M210 M211 M273 M280
        M283 M311 M312 M321 M332 M342 M373 M382 M391 M392 M412 M511 M520
        M530 M540 M620 M650 M781 M904 M905 P444 P446 P510 P517 P525 P526
        P528 P617 P922
        DCN: RA4D8G-K; RA4D8G-T; RA4D8G-U
   *49* G037 G553 H4 H403 H462 H481 H7 H722 H8 J0 J011 J1 J171
        M280 M315 M322 M331 M332 M342 M372 M373 M391 M415 M510 M520 M530
        M541 M781 M904 M905 M910 P444 P446 P510 P517 P525 P526 P528 P617
        P922
        DCN: R01361-K; R01361-T; R01361-U
M2 *50* G037 G553 H4 H402 H461 H481 H7 H721 H8 J0 J011 J1 J171
        J5 J561 M280 M315 M322 M331 M332 M342 M372 M373 M391 M415 M510
        M520 M530 M541 M781 M904 M905 M910 P444 P446 P510 P517 P525 P526
        P528 P617 P922
        DCN: R01449-K; R01449-T; R01449-U; R10058-K; R10058-T; R10058-U
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        M314 M315 M321 M331 M332 M342 M372 M373 M391 M412 M511 M520 M530
        M540 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
        RIN: 01010
        DCN: R03935-K; R03935-T; R03935-U
M2 *52* G032 G033 G060 G630 H100 H181 H4 H402 H403 H461 H481 H483 H720
        H721 H731 H8 J011 J171 M280 M314 M316 M321 M332 M333 M334 M342
        M343 M344 M372 M373 M383 M391 M415 M510 M520 M530 M541 M620 M650
        M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
        DCN: RAEJ8C-K; RAEJ8C-T; RAEJ8C-U
   *53* G037 G553 H4 H402 H461 H481 H7
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                                        H721 H8
        J5 J561 M210 M211 M272 M281 M315 M316 M321 M332 M333 M342 M372
        M373 M391 M415 M510 M520 M530 M541 M781 M904 M905 P444 P446 P510
        P517 P525 P526 P528 P617 P922
        DCN: R21800-K; R21800-T; R21800-U
M2 *54* A111 A960 B615 B702 B713 B720 B795 B815 B833 C710 D011 D019 D931
        F012 F013 F014 F015 F113 H1 H100 H122 H2 H201 H4 H402 H422
        H8 L943 M280 M311 M321 M342 M373 M391 M411 M511 M521 M530 M540
        M630 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
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DCN: R18980-K; R18980-T; R18980-U
M2 *55* D021 D601 H1 H102 H181 H4 H401 H481 H5 H541 H8 M210 M213
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        M540 M781 M904 M905 M910 P444 P446 P510 P517 P525 P526 P528 P617
        P922
        DCN: R01969-K; R01969-T; R01969-U
   *56* F011 F013 F014 F433 G013 G017 G100 H1
                                             H100 H141 H181 H2
        H5 H521 H542 H6 H601 H602 H642 H8 J0 J011 J3 J321 M1
        M123 M136 M210 M211 M272 M282 M313 M321 M332 M342 M383 M391 M413
        M510 M521 M532 M540 M781 M904 M905 P444 P446 P510 P517 P525 P526
        P528 P617 P922
        DCN: RAOGIN-K; RAOGIN-T; RAOGIN-U
M2 *57* F012 F522 G012 G013 G100 H103 H141 H401 H441 K431 K432 M121 M143
        M210 M211 M240 M271 M281 M311 M320 M321 M342 M373 M391 M413 M510
        M520 M521 M530 M532 M540 M620 M650 M781 M904 M905 P444 P446 P510
        P517 P525 P526 P528 P617 P922
        DCN: RAOM8Z-K; RAOM8Z-T; RAOM8Z-U
M2 *58* F011 F012 F013 F423 G010 G017 G100 H1 H102 H141 H181 H2
        H5 H541 H6 H602 H641 H8 J0 J011 J3 J321 M1 M123 M136
        M210 M211 M240 M272 M273 M281 M311 M321 M342 M373 M391 M413 M510
        M521 M532 M540 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528
        P617 P922
        DCN: RA61W6-K; RA61W6-T; RA61W6-U
M2 *59* D011 D022 E240 F011 F014 F553 H1 H121 H181 H2 H202 H6 H602
        H641 L943 M210 M211 M273 M281 M320 M412 M511 M521 M530 M540 M781
        M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
        RIN: 03676
        DCN: R22668-K; R22668-T; R22668-U
   *60* D013 D022 D601 F011 F012 F014 F019 F433 F523 G013 G100 H1 H141
M2
        M116 M280 M312 M321 M332 M342 M383 M391 M412 M511 M522 M531 M540
        M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
        DCN: R20437-K; R20437-T; R20437-U
M2 *61* D011 D012 E850 F011 F014 F553 H1 H121 H181 H2 H202 L943 M210
        M211 M240 M273 M281 M320 M412 M511 M521 M530 M540 M781 M904 M905
        P444 P446 P510 P517 P525 P526 P528 P617 P922
        RIN: 46639
        DCN: RA04JZ-K; RA04JZ-T; RA04JZ-U
  *62* D011 D014 D022 D790 E410 F011 F014 F433 H1 H181 H2 H201 H6
        H601 H641 J5 J521 L9 L941 M1 M116 M210 M211 M240 M281 M312
        M321 M332 M342 M373 M391 M412 M512 M521 M530 M540 M781 M904 M905
        P444 P446 P510 P517 P525 P526 P528 P617 P922
        RIN: 01123 01608
        DCN: RA07U9-K; RA07U9-T; RA07U9-U
   *63* C316 F011 F012 F423 G015 G100 H1 H181 H2 H201 H5
M2
        J0 J011 J3 J331 K0 K3 K353 M210 M211 M212 M272 M273 M281
        M311 M321 M342 M373 M391 M413 M510 M521 M531 M540 M781 M904 M905
        P444 P446 P510 P517 P525 P526 P528 P617 P922
        DCN: R06022-K; R06022-T; R06022-U; R16364-K; R16364-T; R16364-U
M2 *64* D011 D022 E800 H1 H103 H182 H2 H201 H6 H602 H641 M210 M211
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        M781 M904 M905 M910 P444 P446 P510 P517 P525 P526 P528 P617 P922
        DCN: R00026-K; R00026-T; R00026-U; R08400-K; R08400-T; R08400-U
M2 *65* F011 F014 F017 F433 G013 G019 G100 H1 H181 H2 H201 H4 H401
        M332 M342 M381 M391 M413 M510 M521 M532 M540 M781 M904 M905 M910
        P444 P446 P510 P517 P525 P526 P528 P617 P922
        DCN: R00066-K; R00066-T; R00066-U
  *66* D013 D019 D022 D712 D799 F011 F014 F433 H1 H181 H2 H201 H212
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H6 H602 H641 J5 J522 L9 L921 L999 M280 M313 M321 M332 M342

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M383 M391 M412 M512 M521 M530 M540 M781 M904 M905 P444 P446 P510
        P517 P525 P526 P528 P617 P922
        DCN: R06637-K; R06637-T; R06637-U; R14531-K; R14531-T; R14531-U
M2 *67* D011 D022 E800 F011 F014 F553 H1 H183 H2 H203 H6 H685 J0
        J011 J2 J271 M220 M223 M231 M262 M281 M312 M313 M321 M332 M342
        M383 M392 M412 M511 M521 M530 M540 M781 M904 M905 P444 P446 P510
        P517 P525 P526 P528 P617 P922
        DCN: R04431-K; R04431-T; R04431-U
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M2 *68* D014 D740 F011 F014 F433 G013 G100 H1 H181 H2
        H601 H641 J5 J522 J581 L9 L910 M1 M123 M131 M280 M312 M321
        M332 M342 M383 M391 M412 M511 M521 M531 M540 M781 M904 M905 P444
        P446 P510 P517 P525 P526 P528 P617 P922
        DCN: R12049-K; R12049-T; R12049-U
M2 *69* D011 D013 E320 G010 G100 H1 H182 H2 H202 K0 L4 L463 M210
        M211 M273 M282 M311 M322 M342 M373 M392 M412 M511 M520 M531 M540
        M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
        DCN: R17747-K; R17747-T; R17747-U
M2 *70* C017 C100 C800 C801 C803 C804 C805 C806 C807 D013 D023 D740 F011
        F012 F014 F111 F553 H1 H100 H122 H2 H201 H211 H5 H542 H8
           J011 J3 J311 L910 M210 M211 M272 M282 M320 M411 M511 M522
        M530 M540 M640 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528
        P617 P922
        DCN: R07015-K; R07015-T; R07015-U
   *71* D023 E340 H4 H401 H461 H8 J0 J011 J2 J251 M210 M211 M272
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        P517 P525 P526 P528 P617 P922
        DCN: R17247-K; R17247-T; R17247-U
    *72* D011 D014 D932 H1 H181 H2 H201 H211 J5 J522 L9 L910 M210
        M211 M273 M282 M320 M412 M511 M520 M530 M540 M781 M904 M905 M910
        P444 P446 P510 P517 P525 P526 P528 P617 P922
        DCN: R00501-K; R00501-T; R00501-U
    *73* F012 F013 F014 F015 F431 F432 H1 H100 H121 J5 J521 L9 L941
        M1 M116 M280 M320 M413 M510 M522 M530 M540 M781 M904 M905 P444
        P446 P510 P517 P525 P526 P528 P617 P922
        DCN: R11075-K; R11075-T; R11075-U
                                             H181 H2 H201 H4
    *74* D011 D021 D029 D030 E550 G030 G530 H1
M2
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        M520 M530 M541 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528
        P617 P922
        DCN: R07064-K; R07064-T; R07064-U; R15129-K; R15129-T; R15129-U
M2 *75* D011 D021 D029 D030 E550 H1 H181 H2 H201 H4 H402 H421 H441
        H7 H716 H721 H8 J5 J561 M210 M213 M231 M273 M281 M320 M412
        M511 M520 M530 M540 M781 M904 M905 M910 P444 P446 P510 P517 P525
        P526 P528 P617 P922
        DCN: R01274-K; R01274-T; R01274-U; R15119-K; R15119-T; R15119-U
M2 *76* G015 G100 H1 H102 H181 H4 H403 H441 H482 H8 M210 M214 M233
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        M531 M540 M781 M904 M905 M910 P444 P446 P510 P517 P525 P526 P528
         P617 P922
        DCN: R02007-K; R02007-T; R02007-U; R06679-K; R06679-T; R06679-U
M2 *77* G015 G100 H1 H102 H181 H4 H403 H441 H482 H8 M210 M214 M233
        M273 M281 M311 M312 M321 M332 M342 M343 M373 M392 M414 M510 M520
        M531 M540 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617
        DCN: RA1EEJ-K; RA1EEJ-T; RA1EEJ-U
    *78* G016 G100 H1 H102 H181 H4 H403 H442 H481 H8 M210 M213 M232
        M273 M281 M312 M321 M332 M343 M373 M391 M414 M510 M520 M531 M540
         M781 M904 M905 M910 P444 P446 P510 P517 P525 P526 P528 P617 P922
         DCN: R01393-K; R01393-T; R01393-U; R06678-K; R06678-T; R06678-U
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M2 *92* G010 G037 G111 G553 H4 H403 H462 H481 H7 H721 H8 J0 J011

M781 M800 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922 DCN: R04150-K; R04150-T; R04150-U; R07095-K; R07095-T; R07095-U

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             P444 P446 P510 P517 P525 P526 P528 P617 P922
             DCN: RA01PF-K; RA01PF-T; RA01PF-U
    M2 *93* G010 G021 G111 G221 H1 H100 H181 H7 H721 J0 J013 J3 J373
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             M371 M381 M391 M392 M414 M510 M520 M532 M540 M781 M904 M905 P444
             P446 P510 P517 P525 P526 P528 P617 P922
             DCN: RA1459-K; RA1459-T; RA1459-U
    M2 *94* F011 F012 F014 F015 F019 F423 F499 F521 H2 H211 J0 J013 J3
             J312 J371 J5 J521 L9 L941 M280 M312 M321 M332 M343 M349 M371
             M391 M413 M510 M523 M530 M540 M781 M904 M905 M910 P444 P446 P510
             P517 P525 P526 P528 P617 P922
             DCN: R02032-K; R02032-T; R02032-U
    M2 *95* D011 D014 D670 G010 G100 H1 H181 H2 H201 J0 J012 J2 J211
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             M531 M540 M781 M904 M905 M910 P444 P446 P510 P517 P525 P526 P528
             P617 P922
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    M2
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             P444 P446 P510 P517 P525 P526 P528 P617 P922
             DCN: R17080-K; R17080-T; R17080-U
        *97* F011 F012 F013 F014 F433 F541 H1 H101 H123 H2 H201 K0 K7
    M2
             K742 L9 L910 M280 M320 M413 M510 M522 M530 M540 M781 M904 M905
             P444 P446 P510 P517 P525 P526 P528 P617 P922
             DCN: R04592-K; R04592-T; R04592-U; R10186-K; R10186-T; R10186-U
        *98* C316 F012 F431 G013 G015 G100 H4 H401 H441 H8 J0 J011 J1
     M2
             J131 K0 K3 K353 K5 K534 L943 M1 M121 M123 M145 M147 M280
             M320 M413 M510 M521 M532 M540 M781 M904 M905 P444 P446 P510 P517
             P525 P526 P528 P617 P922
             DCN: R12996-K; R12996-T; R12996-U
    M2 *99* G010 G100 H1 H100 H181 M280 M313 M321 M331 M342 M373 M391 M414
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             P528 P617 P922
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L221 ANSWER 24 OF 24 WPIX COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2003-341309 [32] WPIX
DOC. NO. CPI:
                    C2003-089517
                     New orthomolecular vitamin E derivatives useful for the
TITLE:
                    treatment of e.g. cancer.
DERWENT CLASS: B02 D21 E13 INVENTOR(S): WILBURN, M D
PATENT ASSIGNEE(S): (WILB-I) WILBURN M D
COUNTRY COUNT:
PATENT INFORMATION:
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APPLICATION DETAILS:

PATENT NO KIND APPLICATION DATE

US 2003007961 A1 US 2001-886472 20010622

PATENT NO KIND DATE WEEK LA PG MAIN IPC

PRIORITY APPLN. INFO: US 2001-886472 20010622

US 2003007961 A1 20030109 (200332)*

28 A61K038-44

INT. PATENT CLASSIF.:

MAIN:

A61K038-44

SECONDARY:

A61K031-714; A61K031-726

BASIC ABSTRACT:

US2003007961 A UPAB: 20030522

NOVELTY - Orthomolecular vitamin E derivatives (I) are new.

DETAILED DESCRIPTION - Orthomolecular vitamin E derivatives of formula (I), their salts, esters and solvates are new.

dotted line = optional double bond;

A, B, D, E = H or methyl;

R =reaction product derived from Q1, Q2 or phenyl (optionally substituted by 1-5 Q3);

Q1 = e.g. (flava-3-ol)n, alpha -ketoglutaric acid, alanine, flavin coenzymes (such as flavin mononucleotide or flavin adenine dinucleotide), para-amino benzoic acid (PABA) or zeaxanthin; n = 1-12;

Q2 = 1-30C alkyl, 2-30C alkenyl or 2-30C alkynyl (all optionally substituted by 1-12 OH, carboxy, amino, halo, nitro, sulfhydryl or J);

J = phenyl or 5-7 membered heterocyclic ring (containing at least one O, N or S) (both optionally substituted by 1-5 OH, carboxy, halo, nitro, amino, sulfhydryl, methyl, 2-10C alkyl, 2-10C alkenyl, 2-10C alkynyl, methoxy, 2-8C alkoxy or -OC(O)R2 (all optionally substituted, and in which at least one C of alkyl, alkenyl or alkynyl is optionally replaced by N, O or S));

R2 = trifluoromethyl, methyl, 1-10C alkyl, 2-10C alkenyl or 2-10C alkynyl (in which at least one C of alkyl, alkenyl or alkynyl is optionally replaced by N, O or S);

Q3 = OH, carboxy, amino, halo, nitro, sulfhydryl, trifluoromethyl, methyl, 1-8C alkyl, 2-8C alkenyl, 2-8C alkynyl or 2-8C alkoxy (all optionally substituted, and in which at least one C of alkyl, alkenyl or alkynyl is optionally replaced by N, O or S).

The stereochemistry at each of the 2', 4' and 8' positions is R or S. Full definitions are given in the DEFINITIONS (Full Definitions) field.

ACTIVITY - Analgesic; Ophthalmological; Antiinflammatory; Cytostatic; Anorectic; Tranquilizer; Antidepressant; Nootropic; Neuroprotective; Antiparkinsonian; Hepatotropic; Antialcoholic; Cardiant; Antiarthritic; Osteopathic; Antirheumatic; Immunosuppressive; Dermatological; Vasotropic; Antithyroid; Antipsoriatic; Nephrotropic; Antidiabetic; Cerebroprotective; Anti-HIV; Antiarteriosclerotic; Gastrointestinal; Relaxant; Vasotropic; Antisickling; Respiratory; Anticoagulant; Gynecological; Hemostatic; Antiasthmatic; Antigout; Antianemic.

MECHANISM OF ACTION - Platelet Aggregation Inhibitor; Hydroxymethylglutyryl Coenzyme-A (HMG CoA) Reductase Inhibitor.

USE - For effecting a biological activity in an animal, such as aging, longevity, nerve activity, hematopoiesis, maintenance of blood cells, hepatic activity, nephritic activity, heart and cardiovascular function, pulmonary function, muscular function, cartilage health, bone health, joint health, gastrointestinal function, reproductive system function, vision, immune function, cell membrane integrity, pain and inflammation; for treating and preventing cancer, obesity, anxiety, depression, depression secondary to a chronic disease, Alzheimer's disease, Parkinson's disease, demyelineating disorder, peripheral neuropathy, enhancing mood and behavior, cirrhosis, chronic liver disease, alcoholic liver damage, toxic chemical exposure, NSAID-liver damage, estrogen induced liver problems, bile disorder, environmental chemical hypersensitivity, heart and/or artery disease risk due to elevated blood levels of homocysteine, osteoarthritis, rheumatoid arthritis, fibromyalgia, joint injuries, joint inflammation, joint degeneration, osteoporosis, organ transplant rejection, graft rejection, lupus,

uvetitis, Bechet's disease, Graves disease, Guillain-Barre syndrome, psoriasis, acute dermatomyositis, atopic skin disease, scleroderma, eczema, aplastic anemia, primary cirrhosis, autoimmune hepatitis, ulcerative colitis, Crohn's disease, amyotrophic lateral sclerosis, myasthenia gravis, multiple sclerosis, hepatic syndrome, glomerulonephritis, rheumatoid arthritis and diabetes mellitus; for reducing the risk of Sudden Infant Death Syndrome; for maintaining and effecting neuronal membrane ratios of phosphatidyl choline and cholesterol (all claimed). Also useful for treating e.g. septic shock, chronic fatigue syndrome, cachexia, head trauma, immune senescence, inflammatory bowel disorder, muscular dystrophy, neuropathic pain, nervous insult, peripheral nerve injury, renal failure, retinal ischemia, skin aging, diseases relating to lifespan and proliferative capacity of cells, diseases induced by cellular senescence, oxidative stress, age-related memory impairment, ataxia-telangiectasia syndrome, myocardial infarction, peripheral vasoconstriction, organ dysfunction, platelet consumption and activation, mitral valve pathology associated with acute perioperative pulmonary hypertension, chronic obstructive arterial disease, Raynaud's syndrome, renal artery stenosis, deep vein thrombosis, peripheral arterial occlusion, other blood stream thromboses, alloxan action, free fatty acid induced pancreatitis, abetaliporpoteinemia, spontaneous abortion, infertility, sterility, sexual performance, post-menopausal syndrome, prostaglandin disorders, cataracts, ocular hemorrhage, degenerative retinal damage, retinopathy, endothelial injury, asthma, bronchitis, pneumonia, systemic lupus erythematosus, Zollinger-Ellison syndrome, gout, Batter's syndrome; and useful as dietary supplements.

ADVANTAGE - (I) Enhances activity of tocopherols, tocotrienols and the covalently linked compound in the relevant bio-chemical pathways that affects various conditions such as aging and longevity. (I) Decreases the release of superoxides by human peripheral blood neutrophils; reduces the levels of tumor necrosis factor and interleukin-1; increases antibody titers in blood; reduces total serum LDL-cholesterol, apolipoprotein B, thromboxane A2, platelet factor 4, triglycerides and glucose; and decreases lipoprotein A concentration in blood.

Dwg.0/0

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FILE SEGMENT:
                      CPI
                      AB; GI; DCN
FIELD AVAILABILITY:
                      CPI: B03-A; B03-H; B04-B03A; B06-A01; B06-A03; B06-D09;
MANUAL CODES:
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                           B10-C04E; B10-E02; B14-C01; B14-C03; B14-C09;
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                           E07-D12; E10-A04; E10-A17B; E10-A22G; E10-B02A1;
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                           E10-C04C; E10-C04L2; E10-E02E1; E10-E02F1; E10-E04M1
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     2003-341309 [32]
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         P816 P820 P822 P922 P943
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                  J171 K0 L7
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M3 *15* F012 F013 F014 F015 F019 F113 F542 H4 H402 H421 H481 H5 H521
        H8 J5 J522 K0 L8 L812 L821 L831 L835 L9 L910 M1
        M210 M211 M272 M281 M311 M321 M342 M373 M391 M413 M510 M522 M530
        M540 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444
        P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633
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        P922 P943
        DCN: RAA9FL-T; RAA9FL-Q; RAA9FL-N
M3 *16* G015 G100 H4 H402 H441 H481 H5 H541 H8 J0 J011 J1 J171
        M210 M211 M272 M281 M311 M321 M343 M349 M371 M391 M414 M510 M520
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        P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616
        P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816
        P820 P822 P922 P943
        DCN: R00091-T; R00091-Q; R00091-N
M3 *17* F011 F012 F013 F014 F015 F019 F113 F542 H2 H211 H4 H402 H421
        H481 H5 H521 H8 J5 J522 K0 L8 L812 L821 L831 L834 L9
        L910 M210 M211 M272 M281 M311 M321 M342 M373 M391 M413 M510 M522
        M530 M540 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434
        P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625
        P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820
        P822 P922 P943
        DCN: RAA9FM-T; RAA9FM-Q; RAA9FM-N
M3 *18* D011 D013 D931 F012 F013 F014 F015 F113 H1 H103 H122 H2 H201
        H4 H402 H421 H481 H5 H521 H8 J5 J521 K0 L8 L812 L821
        L831 L834 L9 L910 M210 M211 M272 M273 M281 M282 M311 M321 M342
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        P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
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        P811 P812 P813 P816 P820 P822 P922 P943
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DCN: RAA9FV-T; RAA9FV-Q; RAA9FV-N
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        H481 H5 H521 H8 J5 J522 K0 L8 L812 L821 L831 L834 L9
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        M522 M530 M540 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433
        P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616
        P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816
        P820 P822 P922 P943
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        M521 M530 M540 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433
        P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616
        P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816
        P820 P822 P922 P943
        DCN: RAA9FX-T; RAA9FX-Q; RAA9FX-N
M3 *21* F011 F012 F013 F014 F015 F019 F113 F542 H1 H100 H181 H2
           H403 H422 H481 H8 J0 J011 J1 J171 J5 J522 K0 L8
        L812 L821 L834 L9 L910 M280 M311 M313 M321 M332 M342 M343 M349
        M373 M381 M391 M413 M510 M522 M530 M540 M710 M904 M905 P220 P411
        P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520
        P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731
        P738 P811 P812 P813 P816 P820 P822 P922 P943
        DCN: RAA9FY-T; RAA9FY-Q; RAA9FY-N
M3 *22* H1 H181 J0 J011 J2 J271 K0 L7
                                              L722 M210 M211 M262 M273
        M281 M283 M312 M321 M332 M342 M383 M391 M416 M620 M710 M904 M905
        M910 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451
        P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714
        P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
        DCN: R00058-T; R00058-Q; R00058-N
M3 *23* C216 H7 H716 H723 K0 K2 K224 K4 K442 M210 M213 M231 M271
        M282 M313 M321 M332 M342 M383 M391 M416 M710 M904 M905 P220 P411
        P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520
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        P738 P811 P812 P813 P816 P820 P822 P922 P943
        DCN: R06395-T; R06395-Q; R06395-N
M3 *24* H1 H100 H181 J0 J011 J1 J171 M280 M312 M321 M331 M340 M342
        M349 M381 M391 M416 M620 M710 M904 M905 M910 P220 P411 P420 P421
        P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523
        P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811
        P812 P813 P816 P820 P822 P922 P943
        DCN: R01210-T; R01210-Q; R01210-N; R10414-T; R10414-Q; R10414-N
M3 *25* G030 G038 G530 H1 H100 H161 J0 J011 J1 J151 M280 M320 M415
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        P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528
        P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813
        P816 P820 P822 P922 P943
        DCN: R06983-T; R06983-Q; R06983-N
M3 *26* F011 F015 F521 H1 H100 H182 H2 H201 J0 J012 J1 J171 J3
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        M391 M413 M510 M521 M530 M540 M710 M800 M904 M905 P220 P411 P420
        P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
        P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
        P811 P812 P813 P816 P820 P822 P922 P943
        DCN: R11742-T; R11742-Q; R11742-N
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        M280 M320 M412 M511 M520 M531 M540 M710 M904 M905 P220 P411 P420
        P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
        P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
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        DCN: R06069-T; R06069-Q; R06069-N
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        M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
        P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
        P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
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   *29* H1 H100 H181 J0 J011 J1 J171 K0 L2 L250 M280 M314 M321
М3
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        P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520
        P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731
        P738 P811 P812 P813 P816 P820 P822 P922 P943
        DCN: R01661-T; R01661-Q; R01661-N; R04740-T; R04740-Q; R04740-N
M3 *30* G037 G038 G039 G562 G599 H4 H402 H462 H7 H725 H8 J5 J562
        M1 M126 M135 M210 M211 M240 M283 M316 M321 M333 M342 M415 M510
        M520 M530 M542 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433
        P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616
        P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816
        P820 P822 P922 P943
        DCN: R11112-T; R11112-Q; R11112-N
M3 *31* D012 D013 D940 H1 H100 H121 H4 H402 H482 H8 J5 J521 L9
        L910 M280 M313 M321 M331 M343 M373 M391 M412 M511 M520 M530 M540
        M710 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446
        P448 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646
        P711 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922
        P943
        DCN: RA1717-T; RA1717-Q; RA1717-N
   *32* H1 H181 J0 J011 J1 J171 K0 L7 L722 M210 M211 M273 M283
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        P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519
        P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723
        P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
        DCN: R00829-T; R00829-Q; R00829-N
                                           J011 J1 J171 K0 L7 L722
   *33* H1 H181 H4 H401 H481 H8 J0
        M210 M211 M273 M283 M313 M321 M332 M343 M381 M391 M416 M620 M710
        M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
         P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
         P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
        DCN: R12266-T; R12266-Q; R12266-N
M3 *34* F014 F521 H1 H100 H181 J0 J012 J1
                                               J171 J3
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        M322 M332 M342 M343 M349 M371 M381 M391 M413 M510 M521 M530 M540
         M710 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446
         P448 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646
         P711 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922
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         DCN: R08807-T; R08807-Q; R08807-N
M3 *35* D013 D023 D120 G015 G100 H4 H405 H421 H444 H8 M1 M113 M280
         M320 M412 M511 M520 M531 M540 M710 M904 M905 P220 P411 P420 P421
         P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523
         P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811
         P812 P813 P816 P820 P822 P922 P943
         DCN: R04686-T; R04686-Q; R04686-N
M3 *36* G015 G037 G038 G111 G563 H4 H405 H442 H463 H7 H721 H8
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         M332 M342 M372 M391 M414 M510 M520 M531 M541 M710 M904 M905 P220
         P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519
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M3 *37* H1 H181 H4 H401 H481 H8 K0
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M3 *38* H4 H401 H481 H8 J0 J013 J1 J173 M280 M313 M321 M332 M344
        M349 M381 M391 M416 M620 M710 M904 M905 M910 P220 P411 P420 P421
        P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523
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M3 *39* J0 J011 J1
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        DCN: R00118-T; R00118-Q; R00118-N
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M3 *40* F011 F012 F014 F522 H1 H100 H121 H181 H2
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        M905 M910 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
        P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
        P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
        DCN: R00203-T; R00203-Q; R00203-N
                       J011 J1 J131 M210 M213 M232 M240 M281 M320 M414
M3 *41* G013 G100 J0
        M510 M520 M531 M540 M710 M904 M905 P220 P411 P420 P421 P423 P431
        P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528
        P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813
        P816 P820 P822 P922 P943
        DCN: R16027-T; R16027-Q; R16027-N
M3 *42* D013 D022 D120 G013 G100 H4 H402 H442 H8 J5 J521 M1
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        P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
        P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
        P811 P812 P813 P816 P820 P822 P922 P943
        DCN: RA00TD-T; RA00TD-Q; RA00TD-N
M3 *43* G015 G100 H1 H100 H181 H4
                                     H402 H442 H8
                                                   M280 M312 M321 M332
        M342 M373 M391 M414 M510 M520 M531 M540 M710 M904 M905 M910 P220
        P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519
        P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723
        P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
        DCN: R00053-T; R00053-Q; R00053-N
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                                                         J522 L9
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        P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520
        P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731
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        M320 M412 M511 M520 M531 M540 M710 M904 M905 P220 P411 P420 P421
         P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523
        P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811
         P812 P813 P816 P820 P822 P922 P943
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M3 *46* G015 G100 H1 H102 H181 H4 H403 H442 H481 H8 M210 M211 M273
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        J172 J3
        M343 M349 M373 M381 M391 M412 M511 M520 M531 M540 M710 M904 M905
        M910 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451
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        P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
        DCN: R00183-T; R00183-Q; R00183-N
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        P822 P922 P943
        DCN: R00902-T; R00902-Q; R00902-N; R04891-T; R04891-Q; R04891-N
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        P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526
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        DCN: R01170-T; R01170-Q; R01170-N; R09472-T; R09472-Q; R09472-N
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